41.8
V643

SPECIAL TOXICOLOGY NUMBER

LIBRARY
RECEIVED
JAN 24 1924
U.S. Department of Agriculture

Price Two Shillings Net, Jan. 1924
or 21/- $5.00 per ann. (post free).

THE VETERINARY JOURNAL

A Monthly Review of Veterinary Science.

Editor:

FREDERICK HOBDAY, C.M.G., F.R.C.V.S., F.R.S.E.,
HONORARY VETERINARY SURGEON TO HIS MAJESTY THE KING,
OFFICIER DU MÉRITE AGRICOLE (FRANCE),
CAVALIERE DEI SS. MAURIZIO E LAZZARO (ITALY),
HONORARY MEMBER OF THE AMERICAN VETERINARY ASSOCIATION,
AND FORMERLY PROFESSOR IN THE ROYAL VETERINARY COLLEGE, LONDON.

Sub-Editor:
GLADSTONE MAYALL, M.R.C.V.S.

LONDON:
BAILLIÈRE, TINDALL & COX,
8 Henrietta Street, Covent Garden, W.C. 2.

AGENTS { United States—ALEXANDER ECER, Chicago.
Australia—GORDON & GOTCH, Melbourne, Sidney, Wellington.
Canada—THE MACMILLAN CO., Toronto.

Entered as Second-Class Matter in the U.S. Post Office.

Issue No. 553. Vol. 80, No. 1.

Directors:

LT.-COL. J. W. BRITTLEBANK, C.M.G., M.R.C.V.S., D.V.S.M. (Vict.)
Chairman.

T. F. SPENCER, M.R.C.V.S. — G. S. BLAND, F.A.I.
A. W. WAMSLEY. — W. A. HURST.

Joint Managers:

E. R. PICKERING. — GEO. LOCKWOOD.

HORSE AND CATTLE INSURANCE

BLOODSTOCK.

FLAT RACERS. BROOD MARES. STALLIONS. YEARLINGS. FOALS. HURDLERS. STEEPLECHASERS.

FARM HORSES
IN-FOAL MARES
STALLIONS
TRADE HORSES
HUNTERS
POLO PONIES
PEDIGREE BULLS and HEIFERS

CASTRATION RISKS.
TRANSIT & SHOW RISKS.

Write for Prospectuses and Agency Terms.

XUM

KUM

XUM

"GLANDONES"

is our Name for Pluri-Glandular Tablets.

The following are suggested for treating sterility in dogs. The Orchitic for males and the Ovarian for females; the latter may also be used in retained placenta when it cannot otherwise be removed:—

GLANDONES (Orchitic). Containing Thyroid, Supra-renal, Pituitary and Orchitic Substances.

GLANDONES (Ovarian). Containing Thyroid, Ovarian, Pituitary and Mammary Substances.

4/- per 100

The above can be dispensed in powders suitable for pigs or other animals.

Sera and Vaccines

for the prevention, diagnosis and treatment of

Poultry diseases

in addition to those for use with Animals, are regularly stocked.

WILLOWS, FRANCIS, BUTLER & THOMPSON, Ltd.,

WHOLESALE MANUFACTURING DRUGGISTS,

40 ALDERSGATE STREET, LONDON.

Established 1751.

Telegrams: "FORTY, LONDON." Telephones: CITY 3618, 3619.

CONTENTS

EDITORIAL— PAGE

The Value of "Specialising" in Veterinary Journalism 1

GENERAL ARTICLES—

Chronic Lead Poisoning as Observed in Lead-Mining Districts. By EDWARD MORGAN, F.R.C.V.S., D.V.H. 2

The Bellanding, or Poisoning of Land, by Lead-Mine Refuse. By J. A. GARDNER, M.A., F.I.C. 13

Salt Poisoning in Cattle. By WM. SCOTT, F.R.C.V.S. 19

Some Cases of Poisoning in Cattle and Poultry. By J. F. CRAIG, M.A., M.R.C.V.S., and D. KEHOE, M.R.C.V.S. 26

CLINICAL ARTICLES—

Prussic Acid Poisoning from the Use of Linseed Cake. By Major-Gen. Sir JOHN MOORE, K.C.M.G., C.B., F.R.C.V.S. 33

Sewage Poisoning. By Major-Gen. Sir JOHN MOORE, K.C.M.G., C.B., F.R.C.V.S. 34

Poisoning by Ragwort (*Senecio Jacobæa*). By HARRY P. STANDLEY, M.R.C.V.S. 35

Sulphur Poisoning in a Mare. By RALPH BENNETT, F.R.C.V.S.... ... 37

Cases of Poisoning. By Major-Gen. Sir FREDERICK SMITH, K.C.M.G., C.B., F.R.C.V.S.... 37

Poisoning by Potassium Nitrate. By R. HUDSON, F.R.C.V.S. 39

Poisoning by Horsetail (*Equisetum Arvense*). By R. HUDSON, F.R.C.V.S. 40

Poisoning in Calves by Nascent Hydrocyanic Acid Evolved by Cake in Solution. By G. T. DUNNE, F.R.C.V.S. 40

Lead Poisoning in Calves. By RALPH BENNETT, F.R.C.V.S. 42

Mustard Poisoning. By H. TUDOR HUGHES, B.Sc., F.R.C.V.S. 43

A Case of Lead Poisoning in Cattle. By G. H. GEMMELL, F.I.C. ... 45

Some Toxicological Notes. By G. D. LANDER, D.Sc. 45

Poisoning by Battle's Vermin Killer. By G. D. MARTIN, M.R.C.V.S. ... 46

Carbolic Acid Poisoning in a Yorkshire Terrier. By HUGH BEGG, F.R.C.V.S. 47

Poisoning in the Dog. By H. STAINTON, F.R.C.V.S. 47

Mercurial Poisoning. By B. GORTON, M.R.C.V.S., M.P.S. 49

Poisonous Action of Certain Drugs. By HENRY GRAY, M.R.C.V.S. ... 50

Toxicological Cases. By J. F. D. TUTT, M.R.C.V.S., F.L.S., F.Z.S. ... 52

NOTICES 54

THE
VETERINARY JOURNAL
JANUARY, 1924.

Editorial

THE VALUE OF "SPECIALISING" IN VETERINARY JOURNALISM.

To-day is the age of the specialist—and in the actual study of the medicinal and surgical portion of our profession, also in the treatment of either one or other of its animal patient branches, we have a number of men whose names are well established as consultants; men to whom the general practitioner can turn with a reasonable hope of received a more experienced and expert opinion in some of his difficult, daily problems. Veterinary journalism has not yet, however, a sufficiently enthusiastic following to enable it to support a number of specialist papers, and other than that one may be specially adapted as a general news agency (to keep the practitioner up to date in the current events of the day), and another endeavour to cater especially for the professional needs of the general practitioner, whilst another may deal particularly with laboratory researches, the pecuniary support given by a small profession like ours does not permit any great extension of these three headings.

At times, however, a journal is justified in launching forth from its usual groove, and the support which was accorded to the Comparative Medicine number of January last has been the incentive to the present Toxicological number, in which there is a collection of clinical case notes which will, we hope, freshen up, and add to, our knowledge on the subject of animal poisons, and, incidentally, be of value to both practitioner and student; and we propose to follow up presently with a series of special numbers, the next one to be devoted to subjects connected with State Medicine.

Much valuable clinical material is wasted and lost to the profession for the want of being put on record, *and a man who records nothing for the benefit of his fellow men is as one who reaps but does not sow; for he takes all and gives nothing in return.* The response to the request for toxicological material has been very gratifying and more than enough to fill the pages of one issue, so that those whose contributions are held over until the February issue must not be disappointed.

Pressure on our space also compels us to hold the Supplement containing the next section of Sir Frederick Smith's *History of Veterinary Literature.*

The recording of actual facts, in all clinical matters, does much to help the student and the practitioner. He who does this does great service to his profession.

General Articles

CHRONIC LEAD POISONING AS OBSERVED IN LEAD-MINING DISTRICTS.

By EDWARD MORGAN, F.R.C.V.S., D.V.H.,
Puerto Cabello, Venezuela.

In the northern districts of Cardiganshire, of all forms of poisoning among farm stock, that caused by lead is by far the commonest, this being due to the lead mines which are situated on the hills and valleys in those districts. One repeatedly sees announcements in the county press of the injurious effects of the lead mines upon animal and fish life.

Owing to the insidious manner by which the poison is often introduced into the system in such minute quantities and for such a lengthy period (the result being far more serious than if the poison was taken in larger quantities, as happens in acute lead poisoning when one or a few animals accidently pick up paint, etc.), a whole flock in the lead mining district is often poisoned before the suspicions of the owner are aroused. Lead, being a cumulative poison when taken in small doses, one finds that the case is often hopeless before the services of the practitioner are called for.

The waste product, such as stone, gravel, and slime, which often contains a great amount of lead, sometimes as much as 15·60 per cent., is wheeled aside and tipped together on the adjoining land (which is often utilised as pasturage for stock), where it accumulates in the course of time to the size of small hills.

The water utilised at most mines is well aerated, and being of moorland origin, is rich in nitrogenous matter. It is conveyed from mountain lakes to the mines by means of a shallow surface drain, flowing very slowly along this winding channel for miles over peaty soil until it reaches its destination. In its course from the lake it is continually augmented by surface water, but so slow and calm is the flow that one has difficulty in detecting the direction which it takes.

On account of the winding movement of the drain, rendered necessary in order to negotiate the hills on its course, it has generally an extensive watershed of marshy land, the water in consequence being laden with ammonia derived from the decomposition of vegetable matter. If it runs over peaty soil (usually the case in Cardiganshire), it has the disadvantage of an acid reaction, the acid acting on the pipes by dissolving the lead. This often results in lead poisoning. My reason for referring to this water is obvious, because it is quite possible that the water from this drain might be acting, under

certain conditions, on the lead at the mines to such an extent as to cause a certain amount of lead solution to be formed in the streams and rivers which receive the waste water from these mines.

Throughout its course, from the lake to the mine, this drain (which supplies the mine) contains fish (trout) ; but after leaving the mine one finds that the fish have perished, owing to the presence of lead in the water, this lead having reached the water as the drain passed through the mine.

The lead mines, according to mine regulations, should have sediment pits or catch pits in order to allow solid matter to settle in them and so prevent its escape into the rivers and streams. In the past, not to mention the present, these preventive measures have not been carried out properly, and a large amount of poisonous stuff consequently finds its way into the rivers. A few years ago it was so thick that it could be traced for miles upon the streams from the mines joining the River Teify. When the water reaches the Teify Bog—a flat area of land measuring about seven miles by five miles —the river is seen to wind through this peaty, swampy soil for a distance of about ten miles, and here the solid particles settle down at the bottom of the deep pools until a heavier flood occurs which churns the sediment up again, and the river, overflowing its banks, throws this material beyond the usual confines of the river, to deposit it on the adjoining land, which consists in most places of pasturage for horses, cattle, and sheep, as well as extensive areas reserved for hay. The highest percentage of deaths amongst stock occurs on farms situated on both sides of the river between two stations on the Great Western Railway, viz. Strata Florida and Tregaron, a distance of about seven miles apart, and where some of the best farms in Cardiganshire are situated.

Again, water from refuse heaps during heavy rains following a long period of dry weather, after filtering through these poisonous refuse heaps (where undoubtedly a certain amount of reaction has taken place during exposure to the sun and air), flows over adjoining pastures.

During the time of dry easterly winds, the dried pulverised material, as well as fine sand from these heaps, is blown over the surface of the pastures where it is deposited. It is surprising the long distances over which this material can be carried by strong air currents which convey it in clouds. It is seen quite distinctly on the surface of snow, where it has drifted along for a distance of a mile from the mine.

Gravel or sand from these mines is often carted quite innocently by farmers and others for building purposes and for covering carriage

drives and footwalks, with disastrous effects on the poultry as long as a grain of it remains on the ground or adheres to the outside walls of the buildings. Fowls by picking up particles of gravel to which adheres a quantity of lead are frequently poisoned. Rains also wash away parts of this gravel to adjoining pastures, where cases of poisoning have often been met with. It is not easy to estimate the amount of lead which such gravel contains. Not many years ago, however, one company decided to again pass these waste heaps through the mill, and the yield was so high that it paid them better than to drill for fresh ore underground, as the percentage of lead was so high in the contents of these waste heaps.

How Animals are Poisoned.

Animals may obtain lead in any of the following ways :—

(1) From contaminated pastures where water has carried the lead from the mines and deposited it during floods or overflows, this happening sometimes several miles away from the mines.

(2) By grazing on pastures to which the wind has blown the stuff from refuse heaps of mines.

(3) By grazing on pastures where the water escapes from the mill and dressing floors, as well as where rain-water conveys poisonous substances from the waste heaps.

(4) By drinking contaminated water from drains, streams, or rivers.

(5) Fowls by picking up gravel, containing lead, from carriage drives and side walls. Fowls kept in the vicinity of the mines obtain the lead direct from the waste heaps. Ducks and geese succumb to the poison very soon in these districts owing to their instinct of searching for insects and worms in the soil and muddy pools.

(6) Dogs and cats often suffer when kept in the vicinity of the mines. It is almost impossible to breed them near the lead mines, but fully grown dogs and cats resist matters better than young ones do.

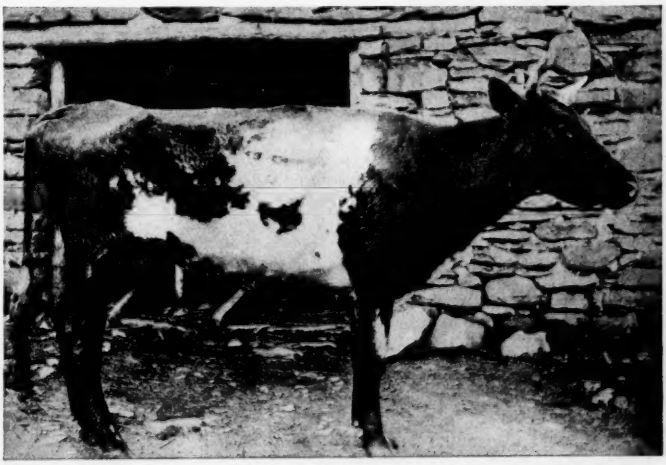
Animals Acquire a Taste for Lead.

In recent years the loss in farm stock has been heaviest amongst horses, especially ponies. The larger number of cases occur as a result of grazing on areas where lead has been deposited during floods. When horses are constantly feeding on contaminated areas, it is useless endeavouring to keep the animals from these areas without a fence. The following cases, being only a few out of many which I have seen in these districts, bears out my opinion respecting the fondness of domesticated animals for the taste of lead :—

XUM

(1) A mare turned out of the stable with her companions into a field about two furlongs from the mine, always immediately left the other animals and went up straight towards the refuse heap to graze what appeared to be coarser grass, which, being contaminated by dust from these refuse heaps at the mines, eventually proved fatal to her.

(2) Sheep belonging to a small holder who kept about twenty ewes on well-cultivated land, abandoned these fields for a common where the pasturage is dry and contains such grass as one finds



PHOTOGRAPH OF COW MANIFESTING SYMPTOMS OF CHRONIC PLUMBISM.

on dry heaths and mountains which have never been cultivated or manured, and where the refuse from the lead mines is deposited with its gravel, slime and stone. The whole surface of this particular pasturage is covered with a thick layer which has been blown from the refuse of these heaps or "tips."

(3) The surface drain or trench, conveying the waste water from a mine passed through a small farm in the district. A mare born and bred on the place could not be enticed to drink any water other than that from the channel containing this polluted water. It was useless offering her any clean water from other sources.

Animals giving milk always succumb to the poison more rapidly than those which are barren. Farmers regard this as one of the predisposing causes. Whether it acts so by lowering the vitality of the

body on account of the foal draining the milk and that anæmia and wasting of fat sets in earlier, or whether it is due to a more rapid absorption of lead from the alimentary canal, remains to be proved.

Through a farm situated at a distance of about a mile from the mine, the drain containing the mine-polluted water passed and overflowed its banks at a certain place, and on this spot horses would continually graze. They could not, however, drink from the leat as this was fenced in. In the course of about twelve years some five or six young horses died from plumbism at this farm, their ages ranging from two to five years. All these were the offspring of the same mare. A most remarkable thing about the latter was, not being bred on the farm, that she never exhibited any symptoms of plumbism, because in the majority of cases animals bred on these lead-contaminated farms very often exhibit far more tolerance to lead than animals from districts free of lead.

Animals bred around these mines seem to have a kind of immunity (or perhaps it would be better to use the term " tolerance ") to the poison. The above is the only case I have met with where an animal, not being bred on a contaminated farm, has been able to resist, where the animals bred on the farm have succumbed, to the poison.

One could give many instances to prove the tolerance of animals bred on these farms around the mines, and also the susceptibility of animals bred in healthy areas when brought to these farms.

(1) A small farm, where the farmer lost fifteen head of stock, including horses and cows, alone within the course of twenty years, all these being animals bought in other districts. He now only keeps animals bred and born on his own farm, and consequently he has not lost any since.

(2) Another adjoining farmer, who has a corner of his land contaminated at certain times of the year, bought two cows last year from a healthy district. Some time later one of these cows died with typical lesions of lead poisoning. The other manifests symptoms of plumbism at present, and is being treated. The rest of his cattle which are home-bred appear quite healthy.

(3) A cow at another farm was bought last year. I was called in to see this cow in June of this year, and found the animal in the last stages of chronic lead poisoning, which were more interesting than usual on account of the Burtonian line (blue line) appearing faintly on the gum, but more pronounced at the interdental papillæ of the incisors. This line is not often seen in animals, unless there is some abnormality of the gums or teeth, such as pus or decayed teeth. Animals do not so often suffer like human beings from septic conditions of the gums and

XUM

teeth. If the animals show suppurative lesions locally around the teeth, the blue line sometimes develops. This has been produced experimentally in animals by inoculation of the gum-tissue with organisms isolated from a case of infective gingivitis in a human being.

(4) Twenty-five head of poultry (hens) were brought to a cottage situated about 500 yards from the rubbish heaps of a lead mine, and within two months they were all dead, while all the others, which were bred on the place, lived. The same applies to ducks and geese which are most susceptible to lead poisoning.

(5) The bed of the River Ystwyth, as already stated, has several feet deep of refuse from the mines, but in certain areas

PHOTOGRAPH OF COW IN LAST STAGES OF CHRONIC PLUMBISM

during the dry season, when the water is low, patches of grass here and there spring up on the wide bed of the river. We had three ewes and lambs which could not be kept away from these areas, in spite of placing them on better pasturage elsewhere. After about four weeks' time the lambs got paralysed in the hind limbs, and the spinal muscles atrophied to a great degree. Eventually spasm of the glottis set in, so they became emaciated and died.

It has been proved that turning cattle on to pasture in mine localities before the dew dries in the morning has most injurious results, the animals exhibiting signs of illness much sooner than when turned on after the disappearance of the dew. We have been investigating into this question in order to try and arrive at some definite explanation

XUM

on this point. As far as we could see, the same amount of poisonous material was adhering to the plants at all hours of the day. The only reasons I can offer are, that by keeping the animals away until the dew disappears in the morning, the animals by going late in the day to these areas of contaminated pasture, having previously been grazing elsewhere on some parts of the farm, would then consume less of the poison with the grass than would have been the case if the stomach had been fairly empty—as is often the case with animals such as the cow, which rest all night. It is known that lead-workers, who partake of a good breakfast before going to the factories, can resist the poison much better than those who commence work without having eaten scarcely any food. The same applies, I think, in the case of cattle, for not only do they eat this lead when the stomach is partly devoid of food in the early morning, but they also consume along with the grass a great amount of dew.

The shepherds around the mines, as soon as they suspect their dogs of eating contaminated food at the lead mines, feed them on plenty of fat.

Absorption of Lead by the Plant. (*Jones Griffith.*)

The question which often arises in connection with the poisoning of stock is whether lead is absorbed by the plant.

Lead enters into the composition of plants growing in affected soils only in very minute quantities. Consequently injurious effects upon animals are likely to be due largely, if not mainly, to the lead in the deposit on the exterior of the plant, or to what may be picked up from the soil itself by close grazing.

A farmer in the locality of one of these mines, whose land was badly contaminated by lead from this mine, lost nearly 200 sheep in one season. Mr. Jones Griffith, at the laboratory of the University College of Wales, analysed the livers of some of these sheep, and found them impregnated with lead to such an extent that a thick precipitate was thrown down in almost every case.

I think the question of poisoning by lead in the localities of the mines is too obvious a fact to be doubted any further (as is often done by lead mine authorities), also that lead ore under certain conditions can be converted into lead solution. Lead in the solid form, such as Galena, is often found in the gastro-intestinal canal of cattle and fowls, and in this form it acts very quickly in the case of fowls. In the case of a three-year-old cow, which died as a result of drinking water from the leat near the mine, where the water was turbid, I found several ounces of lead ore and fine gravel mixed in the stomach.

XUM

The amount of lead taken by animals to cause lead poisoning is difficult to estimate, some animals, as already stated, are far more susceptible than others. The amount of lead in drinking water for human beings need be only very small in order to cause chronic lead poisoning—1/100th of a grain per gallon is sufficient according to Dixon Mann.

Again, it varies with age and condition of the animals, for fully grown animals can tolerate lead better than young animals. Animals bred and born in the localities of the mines have established a certain degree of tolerance towards the metal, i.e. a kind of balance between the intake of the poison and its excretion exists.

Symptoms.—The symptoms vary a little according to the species, age, and quantity of lead consumed—this last item being difficult to estimate. The frequency with which the animal gets access to the poison must also be taken into account—whether it gains access regularly or only at certain periods. It affects the young sooner than the fully grown, and symptoms appear sooner in animals which are working ; muscular exertion seems to hasten matters. The primary symptoms in horses can be best observed when they are at work, because, as a rule, the least abnormality is generally noticed by the horse-man. The same applies to milking cows, for when the milk is unduly diminished in quantity it is sure to be observed. The symptoms are also observed earlier in animals fed indoors than in those fed outside, these latter having to search for their food. For the same reason the first symptoms in ponies which are out all the year may pass unnoticed. These animals may often deceive their owner, for at a glance their thick coat of hair often acts a camouflage, making them appear to be in good condition. A young animal often has the appearance of an old one, with drooping ears, staring coat, and stiff movements. They may be lame either in the hind or fore limbs, or in both, exhibiting dragging movements by their inability to freely extend the joints. They seem to have little control over their limbs, and seem to have a kind of swinging gait. If at work, the animal is easily fatigued. If fed indoors, the animals may be constipated, but this is not a general symptom in horses. Of the domesticated animals, dogs more often suffer from colic, associated with constipation. In cattle there is generally a great deal of interference with digestion, for the animal, being a ruminant, a loss of function in the mechanism of rumination is rather serious, also being sometimes followed by pain in the abdomen, associated, as in the dog, with constipation. Pregnant animals not infrequently abort. The visible mucous membranes are paler than normal. The pupils are dilated ; this may be due, according to

Elsching (quoted by Legge and Goadby), to the direct action of lead upon the unstriped muscular fibre of the vessel walls. Such an action may and does extend to the vessels of the eye muscles, producing paralysis of the muscles of accommodation with dilation of the pupil. I consider the dilation of the pupil, when associated with other symptoms, as being very characteristic in chronic lead poisoning. The pulse rate is accelerated, is hard, and generally about 70. There are always present signs of nervous excitability, as one finds it difficult to approach a previously docile animal ; paralysis of the muscles of accommodation of the eye may be a contributory cause. There may be total or partial blindness, which sometimes comes on gradually. One very characteristic symptom in horses is difficult breathing, which is often called " roaring." This dyspnœa is said to be due to paralysis of the superior laryngeal nerve.

The laboured breathing is sometimes accompanied by fits of coughing. These seizures often come on suddenly when the animal is working either in harness or under saddle. The animal falls down, and after struggling for a long time may eventually manage to rise on its feet. It is quite a common occurrence for a farmer in these districts, on his way to market, to take the harness off the horse and turn the animal into the next field, the animal being unable to proceed on the journey. Perhaps within an hour or so the animal may be able to walk home.

The foals of mares which graze on these contaminated areas are not thriving like other foals. Whether this condition is entirely due to the diminution of milk supply or its poor quality is hard to say, but the milk may contain a certain amount of lead.

The temperature in chronic lead poisoning does not aid in diagnosing a case. It varies from normal to 103° F.

Brain symptoms in cattle are very characteristic of the disease. Farmers in these districts call it " staggers." If the animal is indoors during one of these paroxysms, it attempts even to climb the walls of the buildings, or may attempt to eat parts of the walls or pieces of wood, or presses the head against the wall. If out in a field it turns and falls. The animal often suffers from impaired vision, sometimes total blindness. If the case should be a cow in milk, the yield is considerably diminished, appetite is impaired, and emaciation sets in. The coat is staring, rumination very irregular, and may be altogether absent when the case is at its last stage, while the milk yield may completely cease. The urine is diminished in quantity, and the specific gravity is increased. In the last case the specific gravity was 1037 (in a milch cow the average specific gravity is 1006). The alkalinity of the blood is decreased in lead absorption. Anæmia

XUM

is one of the earliest symptoms in all animals. Basophile granules are sometimes found by making a smear of the blood. This is also present in a few other cases, such as malaria after a prolonged course of arsenic, but this latter refers to man.

In sheep the wool fibres are thin, and there is always a deficiency of yolk. It is always difficult and often impossible to shear them, for they never seem to shed their wool as healthy sheep do. Anæmia is present when sheep are infected with flukes, but in plumbism there might only be a few present, but generally there is a complete absence. Scouring is also absent; there might be a slight attack, but not to such an extent as seen in "liver rot."

Dogs are seized with epileptiform convulsions. The animal runs in wide circles until it drops fatigued, foaming at the mouth. At the onset these attacks come once or twice daily, but eventually they occur more frequently, with the result that the animal becomes emaciated, as well as partially or totally blind. The dog may fall down during one of these attacks over a precipice or into a pool of water and is drowned. Convulsions in lead poisoning are of serious importance; they frequently portend a fatal result.

With regard to cats, they are very susceptible to lead poisoning, the symptoms being more characteristic even than in other animals, owing to the inability of the animal to jump. The muscles of the back and the quadriceps extensor of the hind legs show signs of paralysis.

Fowls seem to be more sensitive to lead than any animals, there being a subacute and a chronic form, some birds surviving only a few days, while other cases prolong for months, the patient eventually dying from exhaustion. The first symptom is the appearance of shell-less eggs. This applies alike to hens, ducks and geese. Brain symptoms follow, with loss of control of movements. The bird may turn like a top, or may wander away for miles, after walking day and night for a week or more. In the subacute form they are unable to pick any food, and are partially or totally blind, being thus unable to follow their companions and unable to reach their perch. The wanderers may pick up a few seeds in the fields, for I have found some in their crops on making a post-mortem examination. They remind one of a rabid animal plodding along, apparently unconscious of all surroundings, or like a "sturdied animal." Another most characteristic symptom in the chronic cases is the peculiar noise they utter, even at night. It is an abnormal cackling noise, as if they had a foreign body in the throat. Professor Newstead, of the Tropical School, Liverpool, informs me that the sparrows around the lead works at Chester suffer in the same way. They are unable to chirp,

but yet attempt, and thus make a peculiar hoarse noise. They are similar to poultry, with their wings ruffled, dirty looking, unable to fly, and in this stage soon fall victims to the cats of Chester.

Post-mortem Appearances, etc.—In poultry sometimes one finds particles of lead in the crop and gizzard, but not so often in the stomachs of other animals, with the exception of cows, which sometimes have the habit of chewing stones on the rubbish heaps or drinking turbid water at the drain near the mine, thus allowing of one finding on post-mortem examination a quantity in the stomach. Inflammation of the abomasum and intestines is rarely seen, except the mucous membrane of the true stomach, which shows small areas of inflammation. The blue line on the gum may be present when gums or teeth are diseased. The lungs are sometimes enlarged. The trachea and bronchi are invariably inflamed and filled with frothy matter or spume. The kidneys, liver, and spleen alike are of a singular blue appearance. The blood remains fluid for a considerable time. Putrefaction sets in most rapidly. There is an absence of omental and kidney fat, and very little orbital fat remains. The peritoneum is thin and glistening. Minute hæmorrhages are found along the intestinal wall. A number of hæmorrhages are found at the base of the brain extending down to the spinal canal.

Upon opening into the abdominal cavity of horses one finds a large amount of straw-coloured fluid, sometimes two gallons or more.

Treatment.—Remove the animals to a fresh pasture which is not contaminated with lead. Horses should not be worked if there are the least symptoms of plumbism present, and they should be disturbed as little as possible. If colic is present, hypodermic injection of atropine should be tried and a purgative given.

The following medicines are antidotes: Acid. Sulph.; Alumen.; Mag. and Sodii. Sulph.; Pot. Iodide. I have used in most cases Belladona, Mag. Sulph., and Pot. Iodide. For poultry a small quantity of Mag. Sulph. is dissolved daily in their drinking water.

In large animals doses of Mag. Sulph. and Pot. Iodide can be mixed with a bran mash. In dogs exhibiting pain in the abdomen pressure can be applied by bandaging, as is done in the case of human beings, to give temporary relief. If the bowels are not relieved enemata must be administered.

THE BELLANDING, OR POISONING OF LAND, BY LEAD-MINE REFUSE.

By J. A. GARDNER, M.A. (Oxon), F.I.C.,
Reader in Biochemistry in the University of London, and Biochemist to St. George's Hospital, S.W.

THE terms "belliam" and "smitham" are dialect words which vary in spelling and somewhat in meaning in different districts. "Smitham" and "smithite" appear to be corruptions of another dialect word, "smeddum." This term is used in the North of England for lead ore in a sufficiently fine state of division to pass through a certain sieve. I believe the size of such particles is approximately that of peas or beans. It is also used in the Midland coal-fields for fine slack, and in other districts for a layer of clay or shale between two beds of coal.

Belliam I am not familiar with, but it is obviously a variation of the common dialect word "bellan" or "belland," probably derived from the Anglo-Saxon "bellan," to bellow or roar. Belland is a name used in Yorkshire, Derbyshire, and Shropshire for fine dust of lead ore. This substance is very poisonous, and so the word has also come to mean the kind of colic produced in man and animals by swallowing particles of lead ore. I think it is a reasonable assumption that poisoning by this means must have been common in the early part of the nineteenth century, for the word in some districts of the North is used as a verb to signify not only lead poisoning but poisoning in general, especially when associated with colic. In some districts it is used to signify the asthma which is a symptom of lead poisoning, and in Cumberland, I believe, it is used for a broken-winded horse. My friend, Professor Hobday, tells me that horses whose roaring is obtained as a result of lead poisoning are, in some districts, known as "bellones." He agrees with me that probably belland and bellones have the same origin.

In *An Old Shropshire Oak*, by Rev. W. J. Waster, vol. i, p. 159, published by Kegan Paul, Trench & Co. in 1886, there is the following quotation from the additions to Camden: "The smoke of the lead produces palsies, consumptions . . . a disorder of the bowels called the *belland*, and affects cattle which feed on the grass or heath contaminated by the smoke. It gives a sweetness to the herbage and makes them eat it greedily; but the proprietors are often forced to pay damages for the cattle which are killed by it."

The lead-mining industry in the United Kingdom appears to have lost its former importance. It was most prosperous about the middle of the nineteenth century, the maximum annual output of 101,997

tons of dressed ore being realised in 1856, when 353 mines were operating. Since 1870 the ouptut has shown a general decline, and in 1914 the total production was only 26,013 tons from about 60 mines.

In the lead-mining region of Shropshire and adjacent portions of Montgomeryshire the lead-bearing veins are contained in Ordovician strata, and trend roughly east and west, or north-west and south-east. The ore-bearing horizon is for practical purposes limited to the Mytton Grits, which immediately overlie the Stiperstones Quartzite at the base of the Ordovician sequence, and are bounded above by the Hope Shales.

The output of lead ore in this district reached a maximum in 1875, when 7,932 tons of concentrate were produced, chiefly from the Roman Gravels, Tankerville and Snailbeach Mines. By far the largest production came from the Snailbeach Mine, which, until recent years, maintained a continuous yearly output, but is now flooded. I gather from the Home Office reports that the output in the whole county in long tons only reached the following insignificant amounts :—

1915	1916	1917	1918	1919
77	32	15	27	12

The Snailbeach Mine is now worked only for barytes (barium sulphate), and the only galena obtained is a by-product in the crushing of this mineral.

Only an insignificant amount of lead ore is mined in a sufficiently pure state for the smelters. Almost invariably the crude ore has to be crushed and subjected to a process of dressing or concentration for removal of waste material, such as quartz, calc spar, fluor spar, country rock, etc. This waste material, which contains only a small percentage of lead, is usually dumped in the neighbourhood of the mine.

Bellanding of Land.

The bellanding of land appears to be a well-known phenomenon to land agents and farmers in mining districts.

Bellanded land may be roughly classified into two types :—

(1) Farm lands, which include old dumps, often partly grassed over, hollows formed by old lead settling ponds, and the like. Here animals may accidentally pick up debris from the banks, or from the sites of the old ponds ; or the rock may gradually weather and the dust be carried down by the rain on to the land. The harmful effect is quite local, and no doubt decreases in danger with time.

(2) Lands in the neighbourhood of dumps in process of formation,

or which are being worked for builders' material or other purposes. Here in dry weather the dust may be carried by the wind, and the land may be more or less bellanded to a considerable distance in the direction of the prevailing wind.

Land of class (1) is not uncommon in parts of Derbyshire and Yorkshire, and in illustration I may cite the experience of farmers on the land adjoining the moorland on which the Grassington Lead Mines are situated. These mines have not been worked for the past sixty or more years, and as far as my recollection goes the farms adjoining the moor include some partially grassed-over dumps, and the farmers have grazing rights over the moorland on which the mines are situated.

My brother, Mr. G. F. H. Gardner, who has a place in the district, a few years ago bought a farm on the edge of the moor by auction, but hearing that the land had not a good reputation he parted with his bargain. I therefore asked him to make some inquiries as to the effect of the lead tips on the land. A farmer who used to farm some of the land in question stated that all animals except those that chewed their cud were affected—horses, sheep, dogs, poultry. The horses were affected with swellings from which they died, the lambs were born with rickety backs, and the dogs and poultry suffered from a kind of dizziness. He spoke of the various complaints as being "ballind."

My brother does not think that at the present time the farmers have much trouble, though two or three years back a horse is said to have died of belland, and before that a dog succumbed, but he states that *it is quite notorious in the district that poultry does not flourish near the lead mines.*

As an example of the second class I may mention a dump or spoil-bank at Snailbeach in Shropshire, which I had occasion to visit some time ago. This bank occupies about nine acres, and fronts the railway on its eastern side for a length of 175 yards. The bank forms a white hillock without any trace of vegetation or animal life. It must have been formed mainly in the palmy days of the local mines, which reached their maximum output about 1875. The bank consists mainly of lumps of calcite, country rock, and quartz, with a filling of more finely divided material. The country rock is for the most part a hard quartzose slightly micaceous schist of greenish grey colour and highly insoluble in acid.

This dump was being worked on a small scale as a source of builders' material. It was separated by a process of washing and screening into lumps of calcite and rock of various grades, and sold for the "rough casting" of walls, concreting and gravelling of paths,

XUM

chicken grit, etc. I had occasion to analyse a number of samples from various parts of the bank, and found the percentage of galena varied from 1·5 to 5, the chalk from 68 to 84, and the country rock from 13 to 26.

At the time of my visit, about the middle of a rather prolonged drought, the surface of the bank was ankle deep in fine dust which, when disturbed, gave a cloud of dust which remained suspended in the air. There was no wind on the day in question, but had a stiff breeze sprung up I have no doubt that a considerable amount of dust would have been carried away by the breeze. This dry dust was only on the surface, and in spite of dry weather there seemed to be plenty of water, for on digging down a foot below the surface the stuff was quite moist. In dry, windy weather a considerable amount of dust would be carried from the bank and distributed over the land in the direction of the wind. In wet and damp weather this would not take place to any extent. However, dust once distributed over the land being insoluble would remain there. In order to gain some idea of the composition of this fine dust carried by wind, I subjected specimens of the surface material to a blast of air in the laboratory and analysed the fine dust carried to the end of the apparatus. To quote one experiment, a sample with a galena content of 4 per cent. yielded a fine dust containing nearly double this percentage. The lead in all the samples collected was in a form (galena) insoluble in water even when well aerated. No lead could be detected in solution after standing with soft water for several days. Evidently, therefore, there is no fear of water being contaminated with lead from such a bank, except in so far as spoil might be carried downstream in suspension.

On leaving a sample in the incubator at body temperature with the gastric juice of a cow, distinct traces of lead were found to have been dissolved.

As far as the farm lands on the western side of the railway are concerned, there is no question of animals picking up débris from the bank, or from the sites of old settling ponds and the like. The bellanding on these lands must be due to dust carried by wind over the land. This undoubtedly takes place under suitable conditions of wind and weather.

A farmer stated that on certain portions of his farm he was unable to graze horses, and that on other parts cattle were affected. He also stated further that cattle, etc., are fond of bellanded land, and seemed to suggest that they liked the flavour of the dust. If this statement is correct, I think the probable explanation is that in this particular district the land needs lime, and that the finely powdered

calcite blown over the surface acts as a lime manure and improves, and perhaps even modifies to some extent, the character of the vegetation. If this is correct, the horses and cattle would naturally seek out these parts of the pasture.

Effects of Chronic Lead Poisoning.

The symptoms vary greatly in different cases according to the character of the animal and the quantity of the poison. The symptoms may be conveniently divided into groups, but it is to be noted that many of these symptoms appear to be closely interconnected, and in many cases it is impossible to decide whether or not a set of symptoms is due to direct action on a single organ or the simultaneous disease of several.

(1) Mouth, stomach, and digestion often give early indications. Patients show loss of appetite, nausea, constipation, wasting, metallic taste in mouth, fœtid breath, and a blue line along the margin of the teeth where they enter the gum. In animals constipation is commonly a symptom, but in some cases the opposite, diarrhœa, has been noticed.

(2) Another early symptom is anæmia. It is often accompanied by jaundice and highly pigmented urine.

(3) One of the commonest symptoms is lead colic.

(4) Another result is paralysis, and is usually limited to the extensors of the fore legs. In human subjects it usually begins in the middle and ring fingers, which cannot be extended. It then goes to the little finger, thumb, and wrist, and we get the well-known phenomenon of wrist-drop. Muscles atropy rapidly. In the horse laryngeal muscles are affected and we get roaring. The external rectus of the eye and muscles of the leg are also affected. In rabbits the hind legs are often paralysed. Young children are also affected in the same way.

(5) Another common symptom is arthralgia, or joint pains.

(6) Under the term saturnine encephalopathia, a number of disorders of the brain may be classed together—headache, giddiness, stupor, tremor and weakness, mania, delirium and convulsions like epilepsy. In some animals these cerebral symptoms are most readily induced.

(7) In predisposed individuals kidney disease, gout, and a state of arterio-sclerosis are induced.

It is very easy to get general vague statements from farmers and other occupants of bellanded land as to the injuries to stock, but I find it very difficult to find cases that have been treated by properly qualified veterinary surgeons, or on which post-mortem examinations

XUM

and toxicological examinations have been made by qualified persons. This is probably due to the facts that (1) farmers as a class are notoriously unwilling to call in and pay the fees of qualified veterinary surgeons except in special cases, and are apt to employ home remedies and call in the help of unqualified men ; (2) the properties of bellanded land are well known to the occupiers. Under some conditions the bellanding is slight, except on very special and rare occasions, and the injurious effects are slow in making their appearances. The farmers can therefore obviate them by not keeping the animals long on the affected pastures.

On inquiry amongst the veterinary surgeons of the Shrewsbury district I was only able to get definite information of one case during more recent years. Mr. Curtis, M.R.C.V.S., who is in practice at Shrewsbury, gave me notes of a bellanded cow which he attended on March 24th, 25th, 28th and April 1st in 1905. The most prominent symptoms were asthma and paralysis. He made a post-mortem on April 2nd. He kindly showed me his notes of the case, which were well taken, and I have not the slightest doubt that the cow died of lead poisoning due to eating belland, though no actual analyses were made.

I also wrote to Mr. W. E. Litt, M.R.C.V.S., of St. John's Hill, Shrewsbury, one of the veterinary inspectors of the Ministry of Agriculture and chief veterinary officer of the district. He replied that he had no information on the subject mentioned, and that he only knew by hearsay that cattle and horses do die occasionally in the lead and barytes mining districts in the county, and that it is said the deaths are due to " bellan " or lead poisoning.

On hunting through the veterinary literature during the past seventy or eighty years I have only been able to find an account of one case of lead poisoning due to lead fumes. In the *Veterinarian*, the chief organ of the veterinary profession, for 1855, there is an account of an action—Stevens *v.* Barwell and Another—tried before Mr. Justice Crompton and a special jury, for damages caused to stock by fumes from a lead furnace.

(1) It shows very clearly the distance the poisonous dust may be carried by the wind.

(2) The poisonous dust in this case was pure lead fume, and its action therefore comparatively violent and rapid.

(3) The expert evidence brought forward seemed to me a model of what such evidence should be. The data were collected in a most careful and thorough manner by Mr. Herapath, a very competent toxicological expert of the period.

(4) The symptoms of bellanding are very clearly set forth.

XUM

This type of bellanding cannot now be common, as lead smelting is only carried on in a few places, and precautions against losing valuable fume are taken.

The only other published accounts which appear to me to bear on bellanding are a series of cases of lead poisoning of cattle by bullet spray on rifle ranges, collected by Professor Tuson and published in the *Veterinarian* in 1864 and 1865. In these cases the finely divided metallic lead was ingested by the animals with their food. Lead is only slowly attacked by gastric juice, and the evidence quoted shows how long this can remain in the paunch of the cow before death occurs. The reason why cattle are differently affected to horses, etc., is of course accounted for partly by the fact that horses crop the herbage more closely than the cow, but mainly because of the peculiar anatomy of the latter. In the cow the food is taken into the paunch, and afterwards brought up and chewed as cud before it is taken into the stomach proper.

SALT POISONING IN CATTLE.

By WM. SCOTT, F.R.C.V.S.,
Bridgwater.

It is perhaps possible that few veterinary surgeons have had the good fortune to be brought in contact with a condition akin to the one upon which this report depends, and as our literature portraying the effects of salt upon cattle is meagre, I feel it to be my duty to the profession to record the following data rather fully.

In the summer of 1921 salt works in this neighbourhood polluted a main waterway with their effluent. This waterway served the triple purpose of a land irrigant, a drinking receiver for cattle, and a field boundary.

The summer in question was abnormally dry, consequently the herbage was non-succulent, and the water supply in "pits," ditches, and "rhines" was very low. My clients, whose fields were bounded by this now contaminated waterway, noticed their cattle were beginning to lose flesh and look unthrifty with symptoms of diarrhœa, but not suspecting any specific cause, I was not consulted until a considerably later date.

The following are the notes made after my first examination. The general appearance of the cattle was not good, and there were distinct evidences of malnutrition. Out of a total of 102 cattle, 37 were very emaciated, hidebound, coat staring and rough looking; they lacked energy and walked in a lazy manner. Diarrhœa was

profuse, and the buttocks and tail very dirty and matted with fæces. This latter condition was very striking, for one does not usually associate diarrhœa on rich pasture lands in dry summers where the herbage is more like hay.

In endeavouring to ascertain the cause of what was undoubtedly a gastro-intestinal irritation, I had no difficulty in concluding some common factor was at work, a something contained in the food, the water, or a parasitic infestation.

A careful examination of the fæces and posterior bowel content appeared to rule the latter out of order. The herbage seemed quite normal, and no vegetable irritants were detected. The water from the waterway was sampled and was found to be exceedingly salt, and noting the effluent from the salt works being poured into the stream higher up, I gave it as my opinion that this was the probable root cause of the trouble.

TABLE I.

Weight of Cattle Drinking Normal Water.

Pre-experiment Weight.	Post-experiment Weight 14 Days Later.	Gain.
No. I. 13 cwt. 1 qr. 0 lb.	14 cwt. 0 qr. 0 lb.	84 lb.
No. II. 8 cwt. 0 qr. 0 lb.	8 cwt. 3 qr. 7 lb.	91 lb.

TABLE II.

Weight of Cattle Drinking Water Contaminated with Salt Effluent.

Pre-experiment Weight.	Post-experiment Weight 14 Days Later.	Loss.
No. III. 11 cwt. 3 qr. 2 lb.	10 cwt. 1 qr. 1 lb.	169 lb.
No. IV. 10 cwt. 0 qr. 0 lb.	8 cwt. 1 qr. 7 lb.	189 lb.

I pointed out to my clients that salt in itself was not a poison, but I could conceive, if given in considerable quantity for a long period, it would have a pernicious influence upon the metabolism quite in keeping with the condition found in their animals. Without positive data, however, this was mere hypothesis, and in event of legal proceedings being taken, I suggested an experiment should be carried out. Accordingly four healthy cattle were taken from the herds which had not had access to the salted water supply and weighed

on the official weighbridge. Each animal was allowed the same kind of food. Two were selected as controls and given ordinary tap-water, and two had placed before them water from the brook contaminated by the salt effluent.

Careful notes were recorded in each case, but as the data of the two pairs of animals were almost on a par with each other, I record in successive tables only one pair in full so far as the liquid and solid content consumption is concerned.

It will be noted, if the weight of the two cattle in Table I are taken together, it equals 21 cwt. 1 qr., while the two cattle in Table II weigh 21 cwt. 3 qr. 2 lb.—the difference of 2 qr. 2 lb. only. If now the gain and loss side is looked into, a greater disparity exists, for while the two cattle drinking normal water gained in fourteen days a total of 175 lb.,* the two cattle drinking "salted" water lost the enormous weight of 358 lb. in the same period. This latter figure taken by itself is great, but to arrive at its accurate import I would point out its face value is even greater when one remembers the gain recorded by the control animals, both sets being placed under an identical dietary.

TABLE III.

Solids consumed per Diem.

BEAST NO. II.

Days.	Chaff.	Meal.	Cake.	Hay.
	lb.	lb.	lb.	lb.
1st	16	8	12	24
2nd	16	8	12	24
3rd	16	8	12	24
4th	14	8	12	24
5th	16	8	10	22
6th	16	8	12	24
7th	16	8	14	26
8th	16	6	12	24
9th	16	8	12	24
10th	14	7	10	24
11th	16	6	12	20
12th	14	8	12	26
13th	14	8	10	22
14th	16	9	12	24
Totals ..	216	108	164	332

* The great increase in weight was largely due to the fact that these animals had during the experiment received an abundance of good rich food, prior to which they were grazing out of doors with great shortage of herbage.

XUM

TABLE IV.

Liquids consumed per Diem: Normal Tap Water.

BEAST No. II.

Days.	Gallons.
1st	6½
2nd	7
3rd	6½
4th	7½
5th	6
6th	8
7th	7½
8th	8
9th	8½
10th	6½
11th	6
12th	5½
13th	7½
14th	7½
Total	98½

TABLE V.

Solids consumed per Diem.

BEAST No. IV.

Days.	Chaff.	Meal.	Cake.	Hay.
	lb.	lb.	lb.	lb.
1st	16	8	12	24
2nd	16	7	12	24
3rd	14	6	10	22
4th	nil	nil	4	6
5th	10	4	6	10
6th	7	6	7	10
7th	nil	nil	4	3
8th	nil	nil	nil	10
*9th	nil	nil	2	6
10th	2	1	4	5
11th	6	4	6	6
12th	7	nil	nil	2
13th	2	1	2	1
14th	nil	nil	1	4
Totals ..	80	37	70	133

* NOTE.—On this date the owner gave 6 gallons of fresh water.

TABLE VI.

Liquids consumed per Diem: Salt Water from Effluent Polluted Drain.

BEAST NO. IV.

Days.	Gallons.
1st	½
2nd	1½
3rd	3½
4th	3
5th	5
6th	nil
7th	4½
8th	½
9th	6 (fresh water)
10th	6
11th	3
12th	3½
13th	3½
14th	1
Total	41½

CLINICAL DATA AND SYMPTOMS.

Beast No. II remained normal throughout and can be dispensed with. In the case of beast No. IV, on the 4th day the bowels became relaxed, the appetite was bad, and the temperature rose steadily, reaching a maximum of 105·2° F. on the 9th day (see chart). The pulse increased in beats to 80–90 per minute.

On the 6th day the animal began to cough and discharge at the nose and eyes, at first mucous and later slightly muco-purulent. The coat began to be erect and the hidebound condition became very marked in so short a time. From the 7th day rumination was practically suspended. The abdomen became tucked up and the head was depressed, while the eye became dull and slightly sunken. The mouth and muzzle were dry and hot, and grinding of the teeth was noted.

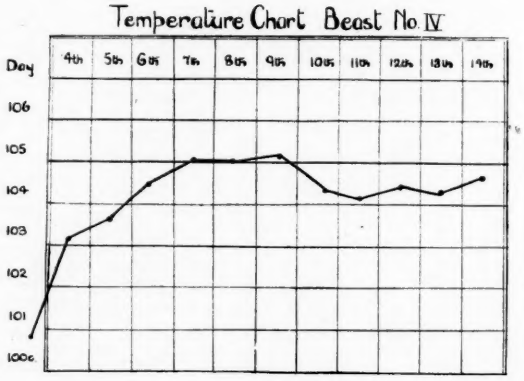
On auscultating the lungs on the 8th day there were noted distinct bronchial râles with peripheral crepitus, and the breathing became hurried. The fæces and their condition deserve special notice.

On the 5th day the bowels were relaxed slightly. On the 7th the evacuation was thin and watery, intermixed with semi-solid lumps. There was considerable gas formation, and the odour emitted was very foul. On the 9th day there was profuse diarrhœa slightly hæmorrhagic, and tenismus for the first time was now noted. All these foregoing phenomena gradually disappeared after the animal was returned to the normal.

Necropsy.—I made a post-mortem of two cows that had died.

XUM

No. 1: This subject was in a very emaciated condition, the buttocks and quarters were matted with fæces, the coat was dry and covered with dandruff. Subcutaneous and internal fat was practically absent, and what was present was watery and soft and of a faint yellow tinge. The pericardial sac was dropsical, the heart was soft and flabby and empty of blood. The lower lobes of both lungs were œdematous, with pronounced hypostasis of the left lung. There were about 4 pints of bloodstained serum in the abdomen. The liver was greatly enlarged and cirrhotic. The spleen was smaller than normal. The kidneys were soft and friable. The fourth stomach showed symptoms of old-standing inflammation with hypertrophy



of its coats. The bowel mucosæ was also inflamed and œdematous. The lymphatic glands through the body were much enlarged and œdematous. The blood looked thin and watery.

No. II: This animal was greatly emaciated, coat dry and covered with dandruff. Heart sac dropsical, thorax dropsical, chronic lobar pneumonia, mediastinal glands tubercular. The rumen and reticulum and abomasum deeply inflamed. The bowels inflamed and shrunken, abdominal fat absent, liver cirrhotic, and kidneys like pulp. The flesh was watery and soapy to the feel and was tinged a pale yellow. The blood was thin and watery and showed a great lack of coagulation power.

Microscopical examination of the blood showed a marked diminution of both leucocytes and red corpuscles.

Abortion.—In all eighteen cows aborted. Out of this number, blood capsules were taken from six of them, and the agglutination test, i.e. *specific abortion,* was applied, but with *negative* results.

Analysis of Water.—I am indebted to Mr. Wood, County Analyst, and Mr. Raymond, his assistant, for permission to take extracts from their very exhaustive and interesting reports following upon their analyses of the water taken from the brook at different places contaminated with the salt effluent to which the cattle had access and also the salt effluent itself.

Samples.				
No. I	.. ..	1,716 parts of sodium chloride	per	100,000
No. II	.. ..	1,705 ,,	,,	,,
No. III	.. ..	1,716 ,,	,,	,,
No. IV	.. ..	1,728 ,,	,,	,,

" The expression ' sodium chloride ' here includes all the chlorides in the water expressed in terms of sodium chloride.

" The normal amount of salt in ordinary water flowing in a river or ditch is from 2 to 7 per 100,000. Well water usually contains about the same amount, but it depends on the strata in which the well is. For example, Weston-super-Mare water formerly contained up to 100 parts per 100,000, but this is most unusual, and the water used to taste salt, it being possible to taste the saltness in a cup of tea.

" Sea water in the Irish Channel contains about 2,702 parts of sodium chloride per 100,000.

" Saline constituents of salt effluent in parts per 100,000 (approximate) :

$CaCO_3$	126·0
$CaSO_4$	299·0
$MgSO_4$	168·0
$MgCl_2$	40·3
NaCl	12960·0 "

Compendium.—Salt in itself is not a poison, but its pernicious influence upon the metabolism if persisted in for a length of time and in sufficient quantum must be an accepted fact. In this case it must be remembered also that the salt content was so high as to make the water unpalatable to the animals which drank it. It was, therefore, only partaken of under desperate conditions. The first pathological phase would be impaired digestion followed by the accumulation of toxins consequent upon the normal metabolic changes of the body (auto-intoxication). As time goes on functional derangement is replaced by organic changes in the vital organs themselves, and when this stage is reached the constitution becomes a physical wreck. Abortion in such cases is not to be wondered at, where the developing fœtus is literally starved to death.

From a forensic point of view the case is not without interest.

XUM

The owners claimed heavy damages for loss sustained, and obtained some £3,000 and costs.

SOME CASES OF POISONING IN CATTLE AND POULTRY.

By J. F. CRAIG, M.A., M.R.C.V.S., *Principal*, and D. KEHOE, M.R.C.V.S., *Professor of Pathology, Veterinary College of Ireland.*

Lead Poisoning in Cattle.

Case I.—The animal affected was a cross-bred shorthorn cow about five years old. She was one of fifty cattle which had been grazing in a field a few miles from Dublin. Within the previous ten to fourteen days the owner had lost three cows, the last death occurring on August 9, 1922. These animals were said to have presented similar symptoms, appearing well until about twelve to twenty-four hours or so before death. They showed loss of appetite, reduced milk supply, and remained quiet until very shortly before death, when symptoms of violent excitement set in. The owner had given them Epsom-salt, but stated that this had no effect upon them, and according to him they presented the same symptoms as this cow before the development of violent nervous symptoms. All the cattle were removed to a neighbouring field on August 10, 1922, and this cow was noted not feeding and " back in her milk " on the morning of the 14th. She was then brought to the College. She was stated to have been brought freshly calved two or three weeks previously. On arrival at the College she was placed in a loose box, and there she remained standing in one position, not inclined to move, expression rather anxious, back very markedly arched, tail depressed and held in between hind limbs, flanks rather hollow, respirations not increased. Eyeballs markedly protruded, vessels of sclerotic injected, pupils dilated. Temperature, 101·8° F. Pulse 48. Animal not feeding. No pain on pressure of abdomen over abomasum or elsewhere. Mucous membrane of mouth pallid, extremities of limbs and base of the horns rather cold.

Cow was given between 12 noon and 1 p.m. 1 lb. sodium sulphate and ½ lb. sodium chloride in about 3 pints of water, and during drenching there were a few slight eructations of gas, and the cow passed a very small quantity of black, somewhat tarry fæces only sufficient to stain the wall behind it down to the floor, and for about one minute afterwards she grunted slightly with each expiration. Shortly afterwards in the afternoon she drank about a bucketful of water.

For the rest of the afternoon and evening she remained standing

in the same attitude and not feeding, and gave very little milk in the evening. She was offered this milk, but refused it, but drank about half a bucketful of water. About 10.30 p.m. no change was noted.

About 7.15 the following morning she was stated by the attendant to be violently excited, bellowing loudly and inclined to charge at him on entering the box. She died about 8 a.m. on August 15, 1922.

A post-mortem examination was made at 3 p.m. on the same day. Condition of animal moderate. Abdomen only slightly tympanitic. Rigor mortis marked in hind limbs, moderate in fore. Eyes not prominent, pupils dilated. Under aspect of tail soiled slightly with greenish liquid fæces. Mucous membrane of gums pallid, and a small quantity of brownish-red liquid exudes from angle of lips. An examination of the blood from one ear was negative in regard to anthrax.

On cutting through the skin subcutaneous vessels found to be congested. Blood fairly fluid and dark red in colour.

Peritoneum.—A few small extravasations present on lower aspect of right posterior sac of rumen.

Spleen.—Pulp, brownish-red.

Liver.—Some of the large bile-ducts prominent under the capsule on posterior surface as yellowish-white stripes running towards the portal fissure. Lower portion, especially near the inferior edge, is cirrhotic, hard and grates under the knife. Cut surface brownish and in part lobulation distinct. Interlobular connective tissue increased in amount and bile-ducts thickened and contain brownish mucoid material and some liver flukes. The gall bladder is filled with yellow fairly fluid bile.

Kidneys, bladder, and uterus appear normal.

Heart.—Extremely numerous subepicardial ecchymoses and petechiæ along the coronary furrows. A few ecchymoses (two on the wall and one on the papillary muscle) and a number of petechiæ under the endocardium of the left ventricle. Myocardium light brown in colour and consistence normal.

Lungs.—Slight hypostatic congestion of left lung.

Bronchi contain some froth and mucous membrane slightly congested.

Trachea contains a little froth. Mucous membrane normal.

Rumen fairly full with contents consisting of grass. Anterior portion of ventral sac near the reticulum shows after removal of the epithelium by scraping slight reddening over an area the size of two hands.

Reticulum presents a slight reddening of a similar nature along

some of the ridges towards their free borders, particularly those ridges near the rumen.

Nothing apparently abnormal was found on removal of the contents of these stomachs in handfuls.

Omasum.—Contents extremely dry and firm. The epithelium comes away with the contents and is black on the free surface and grey on deep aspect, and most of the leaves show congested and reddened patches.

Abomasum.—Folds of mucous membrane reddened, and in addition there are deep red patches and spots along their length. The mucous membrane between them is greyish yellow, but also shows dark red spots and patches, and there are similar small patches on the mucous membranes of the abomasum immediately to the right of the folds. The remainder of the pyloric portion is pinkish yellow.

The abomasum contains a small quantity of semi-liquid brownish material comprising finely comminuted vegetable fibres, and mixed with this some particles of sand and pea-sized pieces of smooth stone, and in addition a few small pieces of hard reddish brick-like material. Towards the omasal opening there is found a piece of material a little larger than a bean, dark outside, with putty-like consistence, and on breaking it across a pinkish surface. It has a putty-like smell of linseed oil.

Tests.—A small portion of the latter material was treated with nitric acid and the solution decanted off. On addition of a solution of potassium iodide to a portion of the latter, a yellow precipitate was thrown down, which was dissolved on heating and come down again in golden yellow scales on cooling.

Sulphuric acid in 25 per cent. solution gave also a white precipitate.

It was concluded that the material was lead and was present in considerable amount.

Intestines.—Contents light brownish in colour. Mucous membrane of the small intestines for a few feet of the terminal portion of the ileum somewhat reddened.

Tongue, pharynx, and œsophagus normal.

The owner afterwards visited the original pasture, and found in a wooded portion under some leaves evidently recently disturbed some suspicious material, of which he brought in a quantity. The greater portion of this was a dry, hard, stone-like pinkish material with a smooth outer surface and that fractured with a rough surface with a thin bluish layer visible, resembling in colour that which was found in the abomasum. In addition, there was a piece that was dry, glossy, and red like dried red paint. It gave the reactions noted above for lead.

The contents of the abomasum, and some of the contents of the other stomachs, a piece of the liver, and the material found in the field were forwarded to the analytical chemist for analysis.

He reported that the material from the abomasum proved to be pieces of dried paint, consisting of red oxide of iron and lead carbonate. In solution in the abomasum there was ·006% lead calculated as metallic lead, and lead was also shown to be present in the contents of the other stomachs and in the liver.

The material found in the field was of the same nature as that found in the abomasum. It appeared later that the material found by the owner in the field was among refuse which had been thrown out some months previously after the erection of loose boxes for horses.

One interesting feature of this case is that four days elapsed after the cows were taken off the pasture on which the lead was found before symptoms of lead poisoning developed in this cow.

CASE II.—The viscera of a cow, comprising the stomachs, intestines, liver, spleen, and heart, were received at the College for examination on July 21, 1922, from Mr. M. F. Murphy, M.R.C.V.S., Oldcastle, Co. Meath.

The following history was forwarded with the organs: "This cow was at grass with other cattle, and had been on the land for three months. About four days previous to this animal's death a bullock died after having shown similar symptoms and within twenty-four hours from their onset.

"This cow was found down on the morning of July 21, 1922, although she was seen in apparent good health on the previous evening. She was in a state of coma and seemed quite blind. The pupils were very much contracted. Temperature 100° F. Pulse very thready. She presented spasmodic contractions of the diaphragm and what appeared to be 'stings' of pain at intervals. On July 22, 1922, after stimulant treatment, she seemed to take some notice of her surroundings, and made attempts to rise, but on July 23rd she died."

Mr. Murphy found on post-mortem examination that "the blood was very black, but clotted well; that there were peculiar mottled spots on the ears, that the lungs were quite normal, but that the trachea showed slight submucous hæmorrhages."

On examination of the viscera forwarded the conditions noted were the following:—The mucosa of the anterior ventral sac of the rumen presented large patches of congestion. These were noticeable after removal of the epithelium, which as a result of decomposition was detached from the mucosa in the first three stomachs, and in greater part came away on the surface of their contents. Similar smaller patches of congestion were present on the mucosa of the

leaves of the omasum. The contents remaining in the first three stomachs consisted of grass and were fairly firm.

In the abomasum there was present a small quantity of finely comminuted vegetable material, and in addition a few lumps, each about the size of a bean, of a grey soft putty-like material. The mucous membrane was thickened and dark-brown red in colour.

The intestines, liver, spleen, and heart showed changes resulting from decomposition, but no other changes were observed in connection with them.

Some of the grey, putty-like material found in the abomasum was dissolved in nitric acid, and the solution obtained gave, on addition of a solution of potassium iodide, a yellow precipitate that dissolved on heating, and on cooling came down again as a precipitate in the form of golden scales. It also gave a white precipitate with sulphuric acid.

The conclusion was therefore arrived at that the material consisted of white lead.

Arsenical Poisoning in Cattle.

Near Mullingar in July 1921 a farmer had thirty three-year-old bullocks in one field on which no previous deaths had occurred during the past thirty years. On the morning of the 11th four of these bullocks were found dead, although they were left apparently quite normal the night before. Two died on the 12th, four died the following morning, and two more were reported ill. The symptoms shown consisted of fœtid diarrhœa, colic, and thirst.

A post-mortem examination was carried out on two of the animals. The lesions common to both were reddening of the mucosa of the anterior ventral sac of the rumen, congestion of the mucous membrane of the abomasum and some parts of the small intestines. The blood formed a firm red clot in the heart cavities, and no anthrax bacilli were found in blood films.

It was suggested that the case might be one of arsenical poisoning, and on discussing the case with the owner some facts came out which are of interest in view of the findings of the analyst.

It appeared that up till that summer the bullocks had been watered constantly from a pond. That summer, owing to the hot, dry weather, the pond had gone dry, and for a fortnight previously a well had to be used which had not been drawn upon for twenty years. This water was drawn up by pumping, and was distributed in tubs. On the 9th, two barrels had been got by the steward and each sawn in two to make the tubs for the purpose of supplying the water to the cattle.

Samples of water from the well and these tubs, and the contents

of the stomachs, were forwarded to the chemist for analysis. The analyst reported the presence of considerable percentages of arsenic in the stomach contents in the tub water, but not in the water from the well.

It was therefore apparent that the tubs made from the barrels had conveyed the poison. They had probably been used previously for holding arsenical sheep-dip.

Poisoning by Common Salt.

The bodies of a young turkey and chicken were received on July 20, 1923, from Miss Julia McKenna, Ballinan. Miss McKenna wrote in explanation: "I had a flock of fourteen exceptionally healthy turkeys from six weeks to two months old. Yesterday morning, on going to feed them, I found two of them dead and the remainder in a very sickly condition. This morning (July 18, 1923) they are nearly all dead. They were chiefly fed on 'Clarendo' and rice with nettles mixed, and sometimes dandelion leaves. The night before they got ill a maid I have, when preparing their food (which was boiled rice) mixed with it some cut up chickweed. I had twenty strong chickens about the same age as the turkeys, and to the best of my knowledge they got a small quantity of the food left by the turkeys. Ten of them are dead, and the remainder, I fear, will die also."

In both the turkey poult and the chicken the lesion were the same—some congestion of the mucous membrane of the duodenum. The crop was full of rice and vegetable material. It was mixed with tap water and filtered. In the filtrate a copious white precipitate was thrown down on adding 5 per cent. silver nitrate solution, this precipitate being soluble in ammonia.

The owner was notified that the losses were due to common salt poisoning, and afterwards replied that on investigation she found from the maid who prepared the food that the rice was made on skim milk. She (the maid) carelessly allowed the rinsing water from a butter-worker which was scrubbed with salt to get mixed up with the skim milk.

Salt Poisoning in Ducks.

On a farm near Dublin ten ducks died within two days, about the middle of February 1921. The symptoms shown previous to death consisted of shivering, fluttering of the wings, and fits with the animals lying on their backs.

Two of the ducks were brought to the College for post-mortem examination. The animals were found to be in excellent condition and their crops filled with whole oats, some pieces of potatoes, and grass. The mucous membrane of the small intestines alone showed any

XUM

change; it was diffusely very dark red, especially in the duodenum. No organisms were found in films made of the heart blood.

Some of the crop contents were mixed with tap water and afterwards filtered through filter-paper. The filtrate gave a copious white precipitate with silver nitrate solution soluble in ammonia.

It was therefore concluded that the cause of the losses was salt poisoning. Later it was learned that shortly before the fatalities among the ducks the troughs for the cattle which contained rock salt had been cleaned out and the material thrown on the land to which the ducks had free access. One drake alone survived. It is also curious to observe that no losses occurred among the fowls which were running with the ducks.

Poisoning of Fowl by Common Salt.

Two dead fowls received from W. J. Flanagan, M.R.C.V.S., Ballinasloe, October 31, 1923.

History.—All the birds, hens, turkeys, and geese were perfectly normal on the 28th. This morning the owner found some of them dead and the others dying. The latter were found to be semicomatose, some of them showing brain symptoms.

The lesions found in the two birds received consisted chiefly of congestion of the mucous membrane of the duodenum.

The filtrate from the crop contents yielded a copious white precipitate with 5 per cent. $AgNO_3$ solution soluble in ammonia.

Phosphorus Poisoning in a Fowl.

In a letter received on October 4, 1923, from one of the Poultry Instructresses of the D.A.T.I. stationed at Dundalk, Co. Louth, it was stated that a great number of the birds in a flock of an owner resident near Dundalk had died in one week early in the summer of this year. At that time the owner would find them lying dead in groups of five and six. The same thing was now happening, and the carcase of a fowl was forwarded for examination.

The carcase received was that of a Leghorn pullet in good condition. There was post-mortem discoloration of the skin. No evidence of diarrhœa. The crop was full and contents comprised pieces of potato and grass, and pieces of some reddish rind (apple or potato rind). The contents of the gizzard consisted of greenish vegetable material with a fair amount of grit and some pieces of reddish rind of potato or apple. These contents gave off a strong characteristic odour best compared with that of matches burning in a box in which they have accidentally become alight, and on coming to remove them gave off whitish clouds. When examined in the dark in a small

glass flask plugged with cotton-wool phosphorescence was clearly observed, especially on shaking the flask, and was accentuated after heating.

The mucous membrane of the duodenum just behind the gizzard was light red in colour. The remainder of it was greyish, while the anterior two-thirds of the mucous membrane of the rest of the intestines was pink. A small amount of the contents of the duodenum and also of the intestines, from a point about 18 inches beyond the gizzard, and extending from there for about 12 inches, gave off the same odour as that of the gizzard contents.

The lungs were dark red, and the vessels near the heart were distended with very dark coagulated blood.

The mucous membrane of the mouth, with the exception of that of the tongue and palate, was congested and reddened.

PHOSPHORUS POISONING IN A HEN.

The body of a white Leghorn hen was received from an owner in Dublin, with the following history: "This is the fifth hen that has died suddenly. They apparently are all right, when suddenly they are seized with an attack of paralysis, lose all power of themselves, and die in about fifteen minutes."

The post-mortem appearances noted were: Comb and wattles blackish-red in colour. No evidence of diarrhœa. Skin of breast and abdomen somewhat reddened, subcutaneous and abdominal fat of a reddish-brown colour. The heart and great veins were distended with dark red blood, the crop contained a small quantity of fat and pieces of potato, and the gizzard was full of grit and broken down vegetable material.

The contents of both crop and gizzard gave off a very strong odour of burning matches, as noted in the previous case, and the gizzard contents gave off white clouds and presented distinct phosphorescence on examination in the dark in a flask plugged with cotton-wool.

Clinical Articles

PRUSSIC ACID POISONING FROM THE USE OF LINSEED CAKE.

BY MAJOR-GENERAL SIR JOHN MOORE, K.C.M.G., C.B., F.R.C.V.S., *Reigate.*

LINSEED cake was largely used in the British Expeditionary Force, France, for the feeding of debility cases, as much as 100 tons per month at one time being consumed. Occasional prussic acid poisoning was encountered, but it was quite easily avoided by boiling the cake. English manufactured cakes from reputable

firms were reliable, but more care had to be exercised in the use of foreign-made cakes. Poisoning is due to the presence in the seeds of a nitrogenous glucocide which, on contact with the digestive juices, undergoes hydrolysis with the formation of prussic acid. The change is really affected by a specific enzyme coexistent in the cake, and it takes place more readily at about the body temperature. When in the ordinary process of manufacture the seeds are crushed and steamed to a temperature of 100° C. the enzyme is destroyed or rendered inactive, no danger is to be apprehended; but in the case of cakes where the seeds have been pressed cold or treated at low temperatures with solvents, the risk is great, particularly perhaps if cakes are afterwards subject to mould.

Veterinary hospitals and convalescent horse depots in France had no ill results after an order for boiling cake was promulgated.

SEWAGE POISONING.

By Major-General Sir John Moore, K.C.M.G., C.B., F.R.C.V.S., *Reigate*.

In the early days of the war, before very strict attention could be paid to a pure and clean water supply to animals of the Expeditionary Force in France, and before the adoption of a water discipline, there were instances of undoubted sewage poisoning from drinking at village ponds; French village ponds are never of the cleanest, and in rainy weather, are usually receptacles of the overflow drainage of neighbouring farmyards. The stench of French farmyards in the early mornings towards the end of the war will be quite familiar to those who served in that theatre of the war.

A gross example of sewage poisoning from drinking the water of village ponds occurred amongst French farm-horses at a small village in Normandy, five or six animals dying so far as my memory serves me. The symptoms were partial coma, paralysis, constipation, and death in from ten to twenty-one days, the appetite during the time commonly remaining well sustained, even when the animal was lying prone on the ground.

The inhabitants blamed poisoning of the ponds by carbide from our mechanical transport lorries, and submitted a claim to our G.H.Q. I inquired into the cause of death personally in conjunction with a French veterinarian, and had no difficulty in determining the real reason. The three ponds were brown and stinking with farmyard washings, and the analyst's report of the samples of water which I brought away for his examination was "badly contaminated with sewage." The French inhabitants were duly warned of the danger. Needless to say the claim was dropped.

POISONING BY RAGWORT (*SENECIO JACOBÆA*).

By HARRY P STANDLEY, M.R.C.V.S.,
Norwich.

THE following cases of poisoning by ragwort, which resulted in the deaths of five horses, are interesting, as poisoning by this plant—although well recognised in South Africa and New Zealand—is not often recorded in this country.

The first case was seen by my assistant about the end of January, who was asked to attend a shire gelding, reported to be colicked. On arrival the horse was showing usual symptoms of colic, with the addition of severe purgation. Peritonitis was diagnosed, sedative treatment was adopted; the animal died, and no post-mortem was made.

Case number two, which occurred about two months later, showed similar symptoms to the above, with the same result. No particular attention was paid to the foregoing cases, they simply being put down to ordinary colic, and would have been forgotten had it not have been for the three following cases, which occurred within three months after.

Number three case I was called into about two or three months after the last case. This mare was taken with slow, dull pains, the mucous membranes were deeply injected, pulse about 60, temperature 104.° At this stage no purgation was present. At the outset the usual remedies were applied, consisting of a full dose of physic arecoline and morphia. This mare, however, did not respond to treatment, and lingered on for just over two weeks, getting gradually weaker, but at times seemed to be going on nicely. Whilst this last case was under treatment another horse was taken ill, showing identical symptoms to the foregoing cases, and whether the third animal was the first to die or this last one I do not quite remember, but rather think the fourth horse died during the illness of the third one. In the third and fourth animals, and also in the fifth to be mentioned later on, in the latter stages during the last two or three days of the illness the brain became affected and the animals were practically mad, so much so that it was impossible during the last twenty-four hours to go near them. Particularly was this so in case number four, the animal knocking the mangers and some of the walls down, and it was at this stage that my assistant and myself suspected poison, and after a very exhaustive inquiry it was found on account of the excessively dry summer of the previous year, the steward on the farm, anxious to make all the hay he could for the coming winter, had shut up rather a bare piece of grass on some very light land for this purpose,

XUM

on which the animals had started to be fed about the middle of the following November. Most of this had been cut into chaff, and even then the horses left a good deal of it. Attention had been drawn to this by one of the team-men, but the only answer he got from the Steward was, that if they would not eat it they must leave it.

The last of these cases which I am going to describe occurred at the beginning of the next summer, and on going over the piece of land which had not been ploughed, I was at once struck with the great quantity of ragwort present, and which, taking into consideration the result of the post-mortem made on the last three cases, was undoubtedly the cause of the whole trouble. It was during the illness of case number three that I suggested an examination should be made of the five remaining horses on the farm. The result of this was to pick out case number four and five, which on closer inspection were found to be a long way from right. Cases number four and five both showed visible mucous membranes to be deeply injected and very yellow colour. Both these animals at this time were feeding well, although the pulse was 80 and over in each case ; the temperatures were about one or two degrees higher than normal. It was only a few days after this that case number four was taken, as above stated. Case number five did not show any serious symptoms until about three weeks after this, although during the whole of this time he was under treatment, but once symptoms of pain set in the animal did not last very long—about two or three days. A post-mortem was made on the last three horses, and the only organ that was affected was the liver, which was tremendously enlarged and exceedingly cirrhotic, and when cut into appeared to be very gritty.

The other two or three horses on the farm did not seem to ail anything, probably from some cause or other they had not eaten so much of the ragwort. From the above very rough description of the cases it will be seen it took nearly three months after the animals had started to be fed on the hay containing the ragwort before the first symptoms of illness were noticed, and over six months before the death of the last animal. This, I take, can only be accounted for by the probability that in some portions of the hay the ragwort was much more plentiful than in others, and also seemed to act very much more quickly, the first two cases being taken and dead under twenty-four hours. Of course, I cannot definitely say that these two horses did die from ragwort poisoning, but in view of the latter sequence of cases, I do not think there can be much doubt about it, and I now regret, of course, a post-mortem examination was not made in each of these cases.

XUM

SULPHUR POISONING IN A MARE.

By RALPH BENNETT, F.R.C.V.S.,
Romford.

On September 28th last a mare was brought to me suffering from slight abdominal pain. On inquiry as to diet, etc., I discovered that the owner had given her nearly a pound of Flowers of Sulphur in a bran mash the night before. He had given a like quantity to another horse at the same time, but this second animal did not suffer any ill effects from this heroic treatment.

The mare in question was admitted to the infirmary and was given small doses of linseed oil together with full doses of Tr. Opii Co and Chlorydine. Stimulants were also given. Intense diarrhœa soon developed, with every symptom of acute enteritis. Pain was intermittent, sometimes being very acute indeed, but with prolonged periods of several hours' duration of freedom from pain. I am inclined to attribute these periods of ease entirely to the large doses of opium and morphia which were given.

The mare gradually became weaker and weaker in spite of treatment, and finally died from exhaustion on October 1st, about eighty-four hours after the first signs of uneasiness were noticed.

A very noticeable feature of this case was the persistent and intense diarrhœa, which nothing seemed to check. The evacuations were very frequent and perfectly liquid.

I think there is sufficient evidence to warrant one in concluding that the animal died as a result of sulphur poisoning, yet it is certainly very remarkable that the other horse, which is said to have received a similar dose of sulphur, developed no diarrhœa, and showed no sign of illness at all. This second animal received an oleaginous purgative, and was kept quiet and warm as preventative measures.

CASES OF POISONING.

By Major-General Sir FREDERICK SMITH, K.C.M.G., C.B., F.R.C.V.S.

The following cases occurred very many years ago; my notes have been lost, so that only an outline from memory can be given.

Arsenical Poisoning, Criminal.—A veterinary officer had got the idea firmly rooted in his mind that most of the internal complaints from which army animals suffered were due to arsenical poisoning! The garrison in which he was serving was a large one, with a central hospital for the sick. At this hospital the various veterinary officers met, and the obsession of "A," as we shall designate him, exposed him to a good deal of ridicule. An "outbreak" of arsenical poisoning followed, the cases occurring not only among the patients under

the care of "A," but to other horses in the hospital. Post-mortem and chemical examination confirmed the confident diagnosis of "A," whose vanity became intolerable. He was carefully watched and finally detected. The confession of his hospital assistant, who administered the arsenic under his instructions, completed the evidence. "A" was, of course, not responsible for his actions. Ridicule, the desire for notoriety, and anxiety to give evidence of extraordinary professional skill and acumen, led him to make certain of his diagnosis by deliberately administering arsenic to his own and other patients! The case is probably unique.

MYLABRIS POISONING.—A hospital subordinate, a good and attentive man, had long observed the golden rule of never dispensing without looking at the label on the bottle. Under the stress of an outbreak of disease, with night after night out of bed, he appears to have forgotten his lesson; one night he dispensed from a bottle, without looking at the label, something which he believed to be Pulv. Ipecacuanha. It so happened that he had previously permitted an unauthorised person to go to the medicine cupboard for something, and this man had moved the bottle of Ipecacuanha and replaced it by one of Mylabris. [At that time in India the Chinese Blistering Fly was used instead of Cantharides.] The replacement was a pure accident, and the bottles were of the same size and shape. Seven horses received their midnight dose of what was believed to be Ipecacuanha, and it is certainly remarkable that the dispenser's eye recognised no difference in colour, for the bottle he dispensed from was that containing the Chinese Blistering Fly! In those days, however, hospital lighting was very primitive. When daylight broke the terrible error was discovered, and I was at once informed of the tragedy. Nothing could be done in the way of treatment beyond demulcent drinks. All the cases exhibited continuous though not very acute abdominal pain and all died. There were no symptoms specially directed to the kidneys or bladder. The Ipecacuanha was being administered in full doses, and each of these unfortunate animals received one ounce of the fly! The condition of the lining membrane of the intestines on post-mortem examination may be imagined.

The greater a man's ignorance the greater his self-confidence; supreme confidence is a valuable asset in youth, but has its obvious disadvantages and limitations.

VEGETABLE POISONING.—The dried leaves of a species of Cypress (*Arbor vitæ*) had been recommended to the head-man of a stable as an excellent thing for horses. The owner of the animals was Lieutenant (the late General) Broadwood, a first-class horsemaster whose stable was a pattern of hygienic management. At this distance of

time I do not remember whether his horses as well as polo ponies received these leaves, but only the ponies were affected. Pony after pony was admitted to hospital suffering from enteritis, and suspicion was naturally aroused. All the animals died within a day or two of each other, and the viscera sent for analysis. This led to the confession of the head-man that he had given the dried leaves of this shrub with the object of improving the condition of the animals. There was no criminal intent. But for the confession the case would have been wrapped in obscurity, as no chemical analysis would have identified the poison. Even now I have no notion of its nature, but it is something remarkably virulent. I forget the precise dose of the dried leaves which was given, but something small; nor do I remember for how long the animals had been receiving it.

NICOTINE POISONING.—I have known many horses poisoned by carelessness in dressing mange cases with a solution of Nicotine. In every instance it was shown that the dilution of this extremely poisonous substance had been left to a subordinate, instead of being effected by the only person who understood the danger and the physiological process of absorption by the skin. The skin of the horse absorbs nicotine as readily and fatally as carbolic acid is absorbed by that of the dog.

POISONING BY POTASSIUM NITRATE.

BY R. HUDSON, F.R.C.V.S.,
Retford.

A CLIENT, late one evening, sought my advice regarding a feeding heifer which was grunting slightly and which, from the symptoms he gave, appeared to have an impaction of the stomach and not very bad.

It was impossible for me to see her that night, so I told him to give her 1 pound Mag. Sulph., and gave him some Ammon. Carb. and Nux Vomica Powders. The following morning I met the owner on the road, and he informed me that the heifer was dead and would be taken to the knacker some distance away, where a post-mortem could not conveniently be made.

Shortly afterwards the foreman on the farm was unwell, and his wife mixed him a teaspoonful of the same sample of Mag. Sulph., and though she failed to get it into solution, he bravely swallowed the lot. Immediately afterwards he was seized with severe abdominal pains and faintness, and remarked that "it was no salts," and on examining the remains found it was saltpetre.

According to his report on the heifer, she was seized a few minutes after the draught was administered with tremblings, staggering, and convulsions, in which latter she died within about half an hour.

XUM

Kaufmann (Finlay Dun) states 6 ounces as a poisonous dose for horses or cattle.

A small two-year-old heifer was given 8 ounces by a shopkeeper, serving it in mistake for cattle salts, and, on finding out the error soon after she had received it, the owner came in for advice. He reported that she appeared all right when he left home. I advised him to give her a tumblerful of whisky, and repeat it in an hour if necessary.

She never showed any ill-effects, and I do not think she had any benefit from the whisky, as about four hours elapsed before she received it.

POISONING BY HORSETAIL (*EQUISETUM ARVENSE*).

By R. HUDSON, F.R.C.V.S.,
Retford.

Synonyms: Scouring-rush, Shavegrass, Marestail, Bottlebrush.

Characters: Stem-jointed, branching at joints, arranged like bottlebrush.

Cattle were affected, and they were grazing on clover, the field being rather bare except where the plant grew. In that part a depression formed across the field, and in it the plant was abundant.

The symptoms shown were diarrhœa, weakness of the hind-quarters, and slightly "tucked up" appearance.

No animals died, and on being removed to another pasture they soon recovered.

I have only seen the plant on clover on this occasion, and think it probable the spores came with the clover seed, which might have been foreign. No doubt the greater moisture in the lower part of the field favoured its growth there.

POISONING IN CALVES BY NASCENT HYDROCYANIC ACID EVOLVED BY CAKE IN SOLUTION.

By G. T. DUNNE, F.R.C.V.S.,
Professor of Therapeutics, the Veterinary College, Irish Free State.

It is now of course a well established fact that certain very valuable food-stuffs contain substances which under suitable conditions will generate hydrocyanic acid in varying quantities, these amounts being sometimes sufficiently large as to set up serious illness in farm stock. Amongst these foods are millet, Java beans, linseed, etc., and it is in connection with linseed that I have noticed the toxic action sometimes rather well marked; in fact, I am almost inclined to believe that the freshly generated acid is more powerful than if administered as the B.P. solution.

In December 1920 I was consulted with regard to the death and

illness of some calves on a fairly large farm, the steward in charge being at a loss to account for the sudden and alarming sickness of a number of the young stock. The history narrated to me was briefly: On the evening of the 8th instant ten calves, average age three months, running on pasture, were brought in as usual, and it was the custom to have ready for them in the calf-house a drink prepared from ground linseed cake and water. This was fed to them in two long troughs, each calf having to fend for itself, those able to drink rapidly probably getting the most.

On this particular evening it was noticed that all the calves, about ten minutes after they had consumed the gruel, commenced to breathe very rapidly, stagger about the shed, heads stretched forward, some of them falling on the ground gasping for breath. One of the latter had at intervals what was described to me as convulsions, and died about fifteen minutes from the onset of symptoms. All the others recovered in the course of a few hours, ate a little hay, and did not exhibit any further symptoms of illness.

The steward was of the opinion the calves had eaten something of a poisonous nature in the field they had just come out of, but careful investigation by him did not throw any light on that probable cause.

On the following evening the nine remaining calves were fed in the same way, and ten minutes afterwards they exhibited symptoms similar to those of the previous evening, another calf dying in the same way. The steward then consulted me, as he could not understand why any illness should occur, considering there had been no departure from the method of feeding practised on the farm for many years. I of course suspected hydrocyanic acid poisoning, and on visiting the farm made a post-mortem examination on the second calf that had died. There was nothing remarkable to take note of in this examination beyond the fact that the lungs were very much congested and the blood all through the carcase much darker than normal. I could not detect any odour of hydrocyanic acid from the undigested contents of the stomach.

On making inquiries as to the source of the cake-meal, I was informed it was part of a fresh supply that had been obtained a couple of days previously, and it had been given to the calves on the two occasions of the illness.

On questioning as to the manner in which the meal drink was prepared, I learned the cause of the whole trouble.

It was the usual practice to mix about 3 pounds of the meal with 6 gallons of *cold* water, half of the mixture being poured into each trough. Just immediately before the calves were allowed to

XUM

drink it 1½ gallons of hot water was added to each amount "to take the cold off it." The heat favoured the action of the ferment on the cyanogenetic glucoside, and as the animals consumed the mixture at once the nascent acid could not escape into the air, its generation taking place in their stomachs.

I was much interested as to the probable amount of acid that would be formed from a definite quantity of the meal, and as there was a possibility of legal proceedings being taken against the firm that supplied the material, I sent a sample to an analyst with a request to ascertain the amount of anhydrous HCN capable of being obtained from it.

I received his report, and learned that he was able to obtain 2·8 grains of acid per pound meal. In bringing this amount into comparison with Ac. Hydrocyan. dil., it would appear that the quantity of acid that would be formed in the 9 gallons of gruel would be a little less than 1 ounce of B.P. solution. This amount divided over ten calves hardly seems sufficient to set up such acute symptoms as were manifested unless the generation of acid in the stomach was exceedingly rapid or that nascent acid is particularly toxic.

In my opinion all the trouble could have been prevented either by making up the gruel with boiling water and so destroying any action of the ferment, or by preventing the calves from drinking it for at least fifteen minutes after it had been made in the customary manner on this farm. I have no definite records to show for what period the acid continues to be formed from the time the ferment first becomes active, but it has been stated that in the case of Java bean meal, if it be maintained under suitable conditions, the formation of acid will be continued for over forty-eight hours.

I asked the steward to prepare another drink for the calves from the meal, requesting him to use the full quantity (9 gallons) of boiling water, and allow the mixture to cool down to a suitable temperature for feeding it; but I am sorry to say he was not willing to risk a sacrifice to science.

LEAD POISONING IN CALVES.

By RALPH BENNETT, F.R.C.V.S.,
Romford.

On June 28, 1922, I was called in to investigate the cause of death of two Jersey calves which had been found dead that morning. Post-mortem examination disclosed acute gastro-enteritis; the abomasum particularly being intensely inflamed.

These were members of a bunch of five Jersey heifers of from one to two months old, kept in the same box and fed together. Of

the remaining three calves, two appeared ill, being dull and listless, and showing evidence of slight loss of muscular control.

I suspected poisoning, very possibly due to lead, and sent parts of the stomachs, bowels, and ingesta to the R.V.C. for analysis. The result of analysis was that lead was present in sufficient quantities to cause death.

On examining the place very thoroughly I found that the calves had licked a considerable amount of paint off the inner surface of the door of the box in which they were placed, and no doubt this was the origin of the fatal dose of lead. A peculiar circumstance of the case was that the door had not been painted for ten or twelve years, and one would not have suspected that such old paint would have been sufficiently attractive for the calves to lick persistently enough to ingest a fatal dose of lead. However, the analysis proved that they had done so.

The remaining three calves were treated with doses of Magnes. Sulph. One died, but the other two recovered; one of these latter never showed any clinical sign of illness, so possibly that one had taken little or none of the paint.

MUSTARD POISONING.

By H. TUDOR HUGHES, B.Sc., F.R.C.V.S.,
Oswestry.

MUSTARD seeds (also called *Brassica alba, Brassica nigra, Sinapis albæ semina et Sinapis nigræ semina*), ground up into powder, may be present as an adulterant in cattle foods, and if in excessive amount give rise to trouble. Such a case has been brought under my notice during the past few days. I append the version of the case as sent to me by the owner.

My client was inclined to think that mustard had been maliciously mixed with the cow's food until my inquiries elicited the information that he had had a bag of a new sort of dairy meal from a friend, which he had mixed with another kind for that day's feeding. I explained to him the possibility of ground mustard seeds being present either as an adulterant or condiment, and advised him to take a sample of each variety of meal, put them in separate basins, moisten with cold water, tie over with paper, and stand on the hob overnight to allow of fermentation, and in the morning to make a small hole in the paper and smell if the odour of oil of mustard were present. Such proved to be the case.

White and black mustard both belong to the family Cruciferæ, and have their habitat in Southern Europe and Asia, and are cultivated in temperate climates. White mustard contains mucilage,

about 25 per cent. of a fixed oil, a glucoside sinalbin and an enzyme myrosin. In the presence of water the inactive glucoside is hydrolysed by the myrosin forming the active oil of mustard.

$$\underset{\text{Sinalbin (inactive)}}{C_{30}H_{42}N_2S_2O_{15}} + \underset{\text{Water}}{H_2O} = \underset{\text{Acrinyl isothiocyanate, or oil of mustard (active)}}{C_7H_7ONCS} + \underset{\text{Dextrose}}{C_6H_{12}O_6} + \underset{\text{Sinapin acid sulphate}}{C_{16}H_{24}NO_5HSO_4}$$

(Dixon's *Pharmacology*.)

Black mustard seeds contain less mucilage, but over 30 per cent. of fixed oil, a crystalline glucoside sinigrin and myrosin. Hydrolysis of the glucoside in the presence of water is produced as before.

$$\underset{\text{Sinigrin (inactive)}}{C_{10}H_{16}NS_2KO_9} + \underset{\text{Water}}{H_2O} = \underset{\text{Allyl isothiocyanate (active)}}{CSNC_3H_5} + \underset{\text{Dextrose}}{C_6H_{12}O_6} + \underset{\text{Potassium hydrogen sulphate}}{KHSO_4}$$

(Dixon.)

This case, in conjunction with its biochemistry, is an instance of those sometimes obscure cases where the toxic substance is not present as such in the foodstuff, but is only formed under conditions favourable to fermentation.

The most dangerous glucosides are those which upon hydrolysis form the most active poisons, e.g. prussic acid (HCN), which is formed from the glucosides present in bitter almonds (the ripe seed of *Prunus anygdalus*), the bark of the wild black cherry (*Pruni virginianæ cortex*), and cherry laurel leaves (*Laurocerasi folia*), e.g. in the case of bitter almonds the seeds contain a crystalline glucoside amygdalin, which is decomposed in the presence of water by an enzyme, emulsin.

$$\underset{\text{Amygdalin}}{C_{20}H_{27}NO_{11}} + \underset{\text{Water}}{2H_2O} = \underset{\text{Dextrose}}{2C_6H_{12}O_6} + \underset{\text{Benzaldehyde}}{C_6H_5 . CHO} + \underset{\text{Hydrocyanic acid}}{HCN}$$

Lander (*Veterinary Toxicology*) makes the observation that the whole seeds of black mustard pass through unaltered in the fæces, and that they may be eaten by the pound without any effect. In cake, however, they may be in the crushed condition.

Owner's Report.

I was feeding the cow on fatting cake, turnips, chaffed hay and bran, and on Monday morning I commenced giving her a small quantity of dairy meal.

On Tuesday evening I increased the quantity of the meal, and an hour after eating this my herdsman told me that the cow was sick. I looked at the feed, and at once asked the man if he had put any mustard in it, as it had this characteristic pungent odour.

The cow had her mouth open, and it was evident that it was

inflamed or burnt and very painful, the tongue hanging out and kiddling, i.e. salivating freely. She was very thirsty. I called in Mr. Hughes, the veterinary surgeon, and he at once asked me about her food. By his instructions I put some of the meal in a basin and covered it up tightly, leaving it until morning. The next morning I put my finger through the paper on the basin, and there was then no doubt whatever about what it contained, as it was in smell exactly like freshly made mustard.

A CASE OF LEAD POISONING IN CATTLE.

By G. H. GEMMELL, F.I.C.,
Professor of Chemistry, Royal (Dick) Veterinary College, Edinburgh.

In June 1919 I received from a farmer in Midlothian the stomach of a cow, and on examination of this found a considerable quantity of lead present therein. The stomach also contained what appeared to be chewed cotton rags. Subsequently I received a quantity of dirty rags, and found these to be saturated with lead paint. On investigation it appeared that a railway company had had the station near this farmer's land repainted, and the workmen had thrown the scrapings of old paint, and the rags used in wiping up the paint, over the fence into a plantation, in a corner of the farmer's field. The cows in the field had access to the plantation, and a number of them died. The claim against the railway company by the farmer was defended, but the Sheriff held the case proved, and gave damages for the value of all the cows poisoned.

SOME TOXICOLOGICAL NOTES.

By G. D. LANDER, D.Sc.,
Formerly Professor of Chemistry and Toxicology at the Royal Veterinary College, London.

Oenanthe crocata (Water Dropwort).—The active principle of this plant is Oenanto-toxin, which Cushny believes to be related pharmacologically to Picro-toxin, the active principle of *Cocculus indicus*. I have had sent to me at various times cases of poisoning by this plant, of which the following is typical:—

Fourteen heifers turned out in a field on Monday, February 16th, and four were found dead on Thursday, the 19th. The hearts appeared to be almost bloodless and the livers were very pale in colour. The ditches in which the plants were growing were part of a large drainage system for low-lying land. Some portions of the *Oenanthe crocata* roots were found in the stomach of each animal. In April and the spring of the year, when the ditches are cleaned out and the mud is thrown up on the margins, the roots of this plant become washed clean by rain, and the white dahlia-like tufts of fleshy

roots prove tempting to grazing cattle. The fleshy, spindle-shaped roots when broken across exude drops of milky juice which turn in a short time into saffron-coloured dots (hence the name "crocata"). It is very toxic, and frequently causes death within three or four hours after being eaten.

Cornus sanguinea (Dogwood).—The following account of poisoning by this plant was sent to me by the late Mr. George Upton, M.R.C.V.S., of Epping:—

The farm where the poisoning has taken place is on the borders of Chingford, and the owner traces the deaths of thirteen animals to this cause. I myself had some yearling heifers which were kept without food for forty-eight hours and were turned into the meadow early in the morning with their stomachs empty, and watched by the owner and myself. They ate the dogwood ravenously, and within twelve hours their stomachs and bowels were distended with gas; colic and salivation set in, and two died within forty-eight hours. The hedge had formed part of an old mansion boundary, and there have been no further deaths since this has been removed. Post-mortem revealed a lot of the branches and berries in the stomach. As far as I know, there is nothing known as to the possible existence in "dogwood" of any specific poison. It appears not unlikely that the events narrated by Mr. Upton illustrate the bad effects of the ravenous eating of coarse indigestible vegetable matter. I have heard of similar results following the eating of common hawthorn by sheep.

POISONING BY BATTLE'S VERMIN KILLER.

By G. D. MARTIN, M.R.C.V.S.,
Westcliffe-on-Sea.

THE patient was a little female Pomeranian, seven years old, weighing about 7 or 8 pounds, to which I was called one morning in a hurry on account of what the owner described as "convulsions."

The owner stated that he was very much troubled with mice in his garage, and that he had carefully baited the place with Battle's Vermin Killer on bread.

He had carefully placed the poisoned pieces under the radiator and near various parts of the engine, as he had observed that the mice congregated in that situation on account of the warmth of the engine. He had, on previous occasions, always been very careful that the dog did not enter the garage, but on that morning he forgot to close the door and the little dog ran inside. One or more of the pieces must have been knocked on to the floor by the mice and been eaten by the dog.

XUM

The symptoms produced were typical of strychnine poisoning. I administered apomorphine followed by morphine, and the symptoms were relieved speedily. With careful feeding and nursing the patient made an excellent and complete recovery.

CARBOLIC ACID POISONING IN A YORKSHIRE TERRIER.

By HUGH BEGG, F.R.C.V.S.,
County Veterinary Inspector of Lanarkshire.

THE owner complained that his Yorkshire terrier was suffering from hæmorrhoids, with evidence of suppuration. He was given a 1 in 50 solution of Lysol, and told to dress the parts daily, using a piece of cotton wool.

Three weeks later it appeared that the Lysol solution was finished, and his wife got a 1 in 50 solution of Carbolic Acid, which was also recommended at the time; and as she thought a little suppuration was coming from the anus, she injected a very small quantity, as she said, about 1 inch within the rectum, by means of a bulb syringe. The quantity so injected was not supposed to be more than one teaspoonful of the solution.

Within a few moments symptoms of poisoning were shown, and professional help was sought without loss of time. The dog was found to be champing its jaws and salivating profusely, to be drunken and unable to stand. The eyes seemed amniotic, having a glassy appearance.

A teaspoonful of turpentine was at once administered in oil, and a solution of sulphate of magnesia injected into the rectum. Some of the solution was also given by the mouth at intervals, and a second dose of turpentine was given about an hour after the first one. The symptoms of poisoning were but little reduced for fully half an hour, but thereafter they became less acute, although exacerbations came on periodically, when one would have thought that death was going to ensue. After forty-five minutes, the symptoms gradually wore off, and in fully one and a half hours from the commencement the animal was resenting the presence of strangers, although not quite normal in conduct. Recovery was complete the same evening.

POISONING IN THE DOG.

By H. STAINTON, F.R.C.V.S.,
Kensington.

ONE of the commonest questions heard in the consulting-room is: "Has my dog been poisoned?" and this irrespective of the symptoms.

There are occasions when it is possible to understand this suspicion of the public mind. Thus, in large cities, an outbreak of epidemic

gastritis among cats may be responsible for wholesale deaths in certain areas. The sudden onset of symptoms followed by speedy death may well lead the residents to suppose that a poisoner is at work.

Bona fide cases do occur, however, and the two following, one malicious and the other accidental, may be of interest.

Case 1.—Urgent 'phone message received early one morning from breeder of bulldogs. Stated that he had found one of his best dogs panting and, he thought, dying. He further gave his emphatic opinion that it was due to poisoning. I advised lump of ordinary washing soda (about the size of a cherry) to be given as an emetic at once.

When I arrived, half an hour later, I found the dog (a very heavy, prize bulldog) panting, blowing, and salivating. I hesitate to say what the pulse was—it was beyond counting.

The emetic had acted, and the ground was smothered in frothy vomit with traces of a brown substance.

In my visiting-case I had three Chloretone capsules (5 grains each) and these were given. The diet for the ensuing forty-eight hours consisted of milk and white of egg, and recovery was complete. The Chloretone was given, as no other sedative was available, but the symptoms abated very quickly, and no other treatment could have given better results.

I was shown some dried pieces of liver sewn together with thread and containing a white powder. This, on analysis, proved to be strychnine. The poison had been thrown over a wall from the street into the yard round which stood the kennels.

Recovery was, no doubt, due to the prompt administration of an emetic, and it may be of interest to some to know that ordinary washing soda can be used in this way. It is available in every household—can be given without delay—it is safe and effective. A further point of interest in this case is that the symptoms were by no means characteristic of strychnine poisoning. There were no muscular spasms beyond those of the chest and thorax.

Case 2.—Another urgent 'phone message—dog suddenly taken ill, think dying. I arrived inside ten minutes and found the dog dead and stiff, and the owner distraught. On investigation, it transpired that the owner, an elderly lady, was taking laxative pills, and that the dog, being constipated, had also received one. The prescription of the pill included strychnine $\frac{1}{6}$ grain, and so no more need be said.

This is not the first case in my experience of fatal poisoning in dogs from human tonic or laxative pills. Such pills often contain strychnine or nux vomica, to which the dog is very susceptible.

XUM

MERCURIAL POISONING.

By B. GORTON, M.R.C.V.S., M.P.S.,
Professor of Anatomy in the Royal Veterinary College, London, N.W.

DURING the summer my attention was drawn accidentally to three cases of mercurial poisoning in cats. My advice was sought by the owner of a thermometer factory regarding the best method of ridding his workshop of rats. Among the means I suggested for destroying these pests were cats and dogs, but the owner remarked that he had tried a cat, or rather three cats, during the last year, and each had died from what was undoubtedly mercurialism. This interested me so much that I sought further particulars, and elucidated the following facts :—

All three cats showed similar symptoms, and one at least was treated by a veterinary surgeon for chronic mercurial poisoning without success. The first sign was a general unthriftiness, but little notice was taken of this. Then partial paralysis of the hind limbs came on, and the animal was in difficulties when it endeavoured to walk. The first cat being somewhat of a favourite, was taken to a practitioner who diagnosed the case as one of mercury poisoning, and treated it in the approved manner; examination of the mouth revealed inflammation of the gums and looseness of the teeth. Continued treatment met with no success, and the animal died. Two other cats which succeeded this one in turn were also affected with the paralysis in the same way a few months after being introduced into the building. Each died from the effects of mercury. Since this no cats have been kept there, and after his experience with these animals the owner did not feel jusified in trying a dog as an exterminator of rats.

Cats were not the only animals to suffer. The rats themselves are not immune to the effects of mercury. They are sometimes found lying dead upon the floor, and generally much distended with gas. This is especially the case after the factory has been closed for a few days. Indeed, it was stated that in more cases than one the distension of the abdomen took place before death, and the rats have been found unable to get back into their quarters. Paralysis also was observed in live rats, which were seen, in the morning, laboriously dragging themselves to their holes.

Thus it is seen that the effect upon the rats was similar to the effect upon the cats. This is to be expected, since it is obvious that both animals were subjected to the same conditions. In such a factory as the one concerned, owing to accidental spilling of the metal during the process of manufacture, the benches and floor become coated with a very thin film. Animals living in such a place are constantly in contact with the film, and the mercury may be absorbed

through the skin or, what is more likely in animals that lick themselves considerably, it is taken in by the mouth.

That possible danger to the people working in these factories is recognised is shown by the fact that the Factory Acts deal with the matter very strictly. The benches have to be covered with asbestos and must be kept clean. The workpeople also are enjoined to wash their hands frequently, to remove any traces of the metal, so that contamination of their food is prevented. The liability to poisoning among the workers is quite small if proper precautions are observed. Indeed, it might be said to be almost non-existent, for men working at this process have continued to do so for many years without suffering harm, as long as they have carried out a few simple rules as to cleanliness.

POISONOUS ACTION OF CERTAIN DRUGS.

By HENRY GRAY, M.R.C.V.S.

Sulphur is not infrequently given in repeated large doses to horses and dogs by people who are ignorant of its dangerous effects. I have observed many examples of its poisonous effects in both species. It is shown by superpurgation, the sulphuretted odour of the evacuations, total loss of appetite, which may last as long as a week in an animal that may ultimately recover, an extremely tucked-up abdomen, and in some cases colicky symptoms. In fatal cases, which are not infrequent in the dog, there is coldness of the surface of the body, running down or infrequent pulse and other signs of cardiac failure, relaxation of the anus, and the giving off the sulphuretted odour wherever the animal goes.

The sulphuretted exhalation from the animal may continue for many days, or even a week or more, after the administration of the drug. In the horse, when there is much emaciation, the anus seems sunk into the pelvis, although it stands out prominently from the surrounding tissues, and the eyeballs are retracted within the orbital fossæ.

What causes death in these cases? Is it due to absorption of the sulphuretted gas, or merely to cardiac failure from superpurgation?

The treatment I have found successful in such cases before cardiac failure has advanced is the repeated administration of chalk in gruel.

Barium Chloride is another agent that is frequently dangerous, and not rarely causes death in a few minutes of its administration by the intravenous method.

I have seen a large cart-horse seized with convulsions and die within five minutes of a 30-grain dose being given by the vein. But before this accident I have frequently given this dose to large cart-horses and even carriage-horses without accident. Probably death in the above case was not due to the quantity but to the rapidity of the intravenous injection. If given by the mouth in too large a dose

repeatedly, or the doses are too closely interspaced, it will set up sypmptoms of gastro-enteritis.

Strychnine is one of the most dangerous drugs to use unless with great circumspection. I have frequently been called into consultation regarding dogs affected with a peculiar nervous affection which I have found to be due to overdosing with strychnine, especially in the form of tincture of nux vomica. In 3-minim doses of this tincture will, after two or three doses, produce physiological effects in small dogs. Even in 2-minim doses given twice a day to a 6 or 7 pound dog show their cumulative effects on the *third* day. Dinner pills containing nux vomica intended for the master or mistress, but often given to the pet dog, creates symptoms of strychnine poisoning. Textbooks on therapeutics, when dealing with strychnine or nux vomica, are not very explicit in detailing the dosage of either of these agents.

When the symptoms are very *acute*, that is, when the animal is forced into the lateral position, where it remains (*toxic zone*), the animal should be chloroformed, and when fully under the anæsthetic receive a $\frac{1}{40}$ to $\frac{1}{10}$ grain of apomorphine hydrochloride subcutaneously. Five minutes after receiving this injection the anæsthetic should be discontinued, so that when the animal commences to recover from its effect it can vomit and empty the stomach safely or without the respiratory muscles being stimulated too much to cause death by the effort to vomit. Morphine in a large dose may be injected after the emptying of the stomach to produce narcosis and therefore to prolong relaxation of the muscles. Of course, the emetic treatment is only advised when the poison has been taken by the mouth. As long as the animal can stand it is in the *safety zone*. Failure of respiration after a prolonged or intense spasmodic attack may be overcome by artificial respiration. I have often seen this after a hypodermic injection of strychnine has been given in a comparatively large dose to induce a powerful physiological effect in cases of optic and other paralyses that have failed to respond to a long expectant treatment and only as a last resource. Personally I have not seen much good from strychnine treatment unless adopted in cases of paralysis of two or three months' standing following distemper. It acts as a spinal stimulant, and is only of benefit when all active disease of the medulla has disappeared and resolution is taking place.

I have had several cases of strychnine poisoning in dogs left at a show all night. In one case I found soot in the buccal pouch, and Dr. Lander obtained $\frac{1}{16}$ grain of strychnine from 1 pound of the stomach contents. The show authorities paid the owner her full claim rather than face an action of law. Contrary to the general idea, dogs dead of strychnine poisoning do not often exhibit retraction of the

XUM

angles of the mouth, erection and retraction of the ears or stiffening or extension of the limbs. In fact, there is mostly no characteristic external appearances to suggest that an animal had died of strychnine poisoning. Retraction of the angles of mouth and other external signs usually attributed to be characteristic of death from strychnine are found in death from the epileptiform convulsions of distemper, true epilepsy, or from rabies.

In another case, in which a dog died in convulsions within thirty minutes of being let out into a back yard, Dr. Clough recovered from the stomach contents $\frac{1}{20}$ grain of strychnine alkaloid.

There are no lesions characteristic of strychnine poisoning, but the serous covering of the viscera frequently shows marked vascularity. One-quarter of grain of the alcoholic extract of nux vomica is toxic to an Aberdeen terrier. What is the toxic dose per pound of the body weight? Strychnine salts are absorbed more rapidly than the pure alkaloid.

A chemist's assistant prescribed Easton's Syrup in m.v. capsules for a small Pomeranian. Twenty minutes after the administration of the first and only dose the animal was dead. This counter prescribing cost a well-known firm of chemists £25 in pre-war days; the claim was paid up without much pressure.

Mortal doses of strychnine are not absorbed from the mucous membrane of the normal urinary bladder, and under such circumstances they produce no effect; they may, however, be absorbed from the inflamed mucous membrane coupled with retention.

Strychnine is the commonest agent with which cats and dogs are maliciously destroyed, usually within thirty minutes of its administration. It should be *proscribed* for the destruction of animals humanely (?), especially when there are so many painless means available. It ought to be made a penal offence to use strychnine for the purpose of destroying cats and dogs.

TOXICOLOGICAL CASES.

By J. F. D. TUTT, M.R.C.V.S., F.L.S., F.Z.S.,
Winchester.

Inorganic Poisons.

Phosphorus.—The cases chiefly encountered have been in pigs, that have become poisoned through rat poison being put down, the rats dying in the vicinity of the sty. The symptoms in the acute state being uneasiness, nausea, vomition of food and mucus, with characteristic phosphorous odour; tenesmus and fœtid diarrhœa. In one case post-mortemed some years ago a portion of a dead poisoned rat was found in the stomach.

In one chronic case, the subject of an action at law about two to three years back, the pig was poisoned by a piece of meat impregnated with the common rat poison paste. Great emaciation, thirst, cough, diarrhœa intermittently were observed, together with slight paralysis of the hind quarters. The pig was ill for six weeks. It was not seen by me until day of death.

In a dog poisoned through eating portions of a poisoned rat, vomition and diarrhœa (both bloodstained) with great thirst was observed. Treatment with sulphate of copper proved unavailing, the dog dying within forty-eight hours of the onset of symptoms.

SULPHUR.—Eight horses undergoing treatment for mange during the late war were poisoned through an N.C.O. giving them an excessive dose on a Saturday night without authority. Three were dead the following morning. The other five recovered. The symptoms noticed were colic, flatulence, and diarrhœa, smelling of sulphuretted hydrogen.

Treatment.—Frequently repeated doses of chlorodyne in starch gruel.

SODIUM NITRATE.—Three cases observed during the past eighteen months. The first was found dead and anthrax was suspected. Post-mortem revealed lumps of the substance in the rumen.

The other two cases occurred within forty-eight hours of each other in one dairy. One cow was found dead.

The second cow when brought into the cowshed to be milked, suddenly appeared to go "mad" (to use the observer's own expression), broke the neck chain, rushed into the adjoining root shed, bit a few pieces out of three or four mangolds, then collapsed and died, the whole occurrence only taking a few minutes.

In each case nitrate of soda was found on post-mortem. In all three cases the pastures had been recently dressed with it as a manure.

COMMON SALT.—Ten pigs were poisoned through salt being added (maliciously?) to the buttermilk sent down with the swill. In the cases observed there were symptoms of abdominal pain, diarrhœa, convulsive movements, and death occurred at varying intervals (forty-eight hours in one case). Post-mortem revealed very acute inflammation of the stomach and intestines. Three recovered.

ARSENIC.—A Gordon setter aged five received the minimum dose of Donovan's solution. The symptoms shown within a few hours of one dose only were vomition, abdominal pain, and slight diarrhœa. The owner fortunately knew what the dog had been given and administered an emetic. Recovery ensued, but weakness of the hind quarters was perceptible until the dog died a few years later. Whether or not this was due to the arsenic it is doubtful to say. The owner naturally affirms to this day that it was. The case is

interesting in view of the smallness of the dose and the fact that only one dose, fortunately, was given.

In another case, a cat was given a measured dose of 1 dram of Arsenic Alba in milk. Beyond violent vomition within a few minutes, no other symptoms were observed, and the cat appeared none the worse for it.

STRYCHNINE.—Cases have been encountered in a horse, foxes, dogs, and cats. In the case of the horse great excitement and tetanic spasms and symptoms of abdominal pain. Post-mortem revealed acute inflammation of the stomach.

In foxes the poison was put down in some cases maliciously, and in other instances for rats.

In dogs, the cause has been the same as in the fox. Vomition, tetanic spasms and death. In one case of a Labrador retriever portions of a dead rat were found in the stomach on post-mortem, and over a grain of strychnine extracted on analysis.

ACORN POISONING.—Contrary to the opinion expressed by Lander (p. 270, *Veterinary Toxicology*), I had, some years ago, a number of pigs poisoned through the ingestion of acorns. Four pigs, each eight months old, were affected and were dead when I reached the farm. Post-mortem revealed large quantities of the acorns, with gastro-enteritis, the stomachs presenting a peculiar "tanned" appearance.

CREOSOTE.—A Labrador retriever accidentally fell into a creosote hurdle-dipping tank, and although rescued immediately by the owner, swallowed sufficient to cause its death. The symptoms were vomition, abdominal pain, and violent diarrhœa and straining. Cold extremities. Death occurred about thirty-six hours from the onset of symptoms.

LEAD POISONING.—A shorthorn bull showed acute symptoms after licking some red lead employed to make a joint in a water-trough in the field. A large dose of Epsom-salt produced uneventful recovery, although the owner had previously administered linseed oil.

NOTICES

All communications should be addressed to 8 Henrietta Street, Covent Garden, London, W.C. 2. Telephone: Gerrard 4646. Telegrams: "Baillière, Rand, London."

Letters for the JOURNAL, literary contributions, reports, notices, books for review, exchanges, new instruments or materials, and all matter for publication (except advertisements) should be addressed to the Editor.

Manuscript—preferably typewritten—should be on one side only of paper marked with full name of author.

BINDING CASES for Volume 79 and any other previous volumes can be obtained from the publishers. Price 2s. 9d. post free.

Copy of advertisements should be in the hands of the publishers—Baillière, Tindall and Cox—not later than the 25th of the month, or if proof is required, not later than 23rd.

Annual Subscription, 21s. ($5.00 U.S.A. currency) post free.